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THE UNIVERSITY OF ALBERTA

THE PSYCHOLOGICAL IMPACT OF DIABETES MELLITUS

A Comparative Study

BY

CATHY KREGER

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH
IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR
THE DEGREE OF

MASTER OF EDUCATION

IN

COUNSELLING PSYCHOLOGY

DEPARTMENT OF EDUCATIONAL PSYCHOLOGY

EDMONTON, ALBERTA

SPRING 1990

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FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled "The Psychological Impact of Diabetes Mellitus. A Comparative Study" submitted by Cathy Kreger in partial fulfillment of the requirements for the degree of Master of Education in Counselling Psychology.

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ABSTRACT

Diabetes mellitus is a serious chronic disorder which has the potential to affect virtually every aspect of life for diabetics. Not only must diabetics make major permanent changes in lifestyle in order to manage their disease condition, but they are also often confronted with the development of serious physical complications calling for major adjustment in their day to day functioning. However, relatively little is known about the psychological impact of diabetes from the perspective of diabetics, and relatively few studies have explored the psychological impact of diabetes across the diabetic lifespan.

The following is a descriptive, exploratory study designed to examine the psychological impact of diabetes from the viewpoint of diabetics over the lifespan. Both Type I and Type II diabetics are explored in this study, with comparisons being made between recently diagnosed (diagnosis less than one year prior), and long duration (diabetic duration 10 or more years) diabetics. In addition, comparisons are made between Type I and Type II with regards to the psychological impact of diabetes.

Among the many findings of this study, a positive relationship was shown to exist between the physical functioning of diabetics and their psychological

functioning. Diabetics who were experiencing physical difficulties or impairment were more likely to indicate they were experiencing psychological or psychosocial disorders. In addition, levels of anxiety and depression were found to be much higher in diabetics who demonstrated symptoms of 'severe' secondary complications when compared with diabetics in which such symptoms were minimal or absent. Retinopathy, in particular, was revealed as having a very detrimental impact upon the total life functioning of affected diabetics. Also, distinct differences were noted between Type I and Type II diabetics in terms of the physical changes that occurred across time, in terms of the psychological changes that occurred across time, and in terms of the influence that metabolic control had on their psychological wellbeing.

Recommendations include the implementation of special counselling, educational, and group programs to assist diabetics in preparing for, and dealing with the onset of secondary complications.

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I. INTRODUCTION

Background

Diabetes mellitus is one of the most common chronic diseases in our society today. It affects over six hundred thousand Canadians (Canadian Diabetes Assocation, 1989) and is a leading cause of death, disability and blindness in North America (Popkin, et al., 1987; Davidson, 1981). Over 98% of the persons with diabetes are over the age of 16 (Platt & Sudovar, 1979), and prevalence of the disease increases substantially with age. Between the ages of 30 and 50, approximately 3-5% of the general population have diabetes. By the age of 60, this percentage increases to 10%, and by the age of 80, diabetes can be found in 16-20% of the general population (Krowlewski & Warram, 1985). At the same time, the proportion of elderly people in our society is increasing, and it has been predicted that by the year 2020, 20% of the general population will be over the age of 65 (Connell, et al., 1988). Therefore, as the life expectancy of individuals in our society increases, we can similarly expect the number of diabetic individuals in our society to increase correspondingly. In addition, diabetes is often considered to be a "sneaky" disease. About half of all cases of diabetes may go unrecognized until symptoms become sufficiently noticeable to make persons seek medical advice (Hamburg, Lipsett, Inoff &

Drash, 1980; Krall, 1978; Sussman & Metz, 1975). Consequently, the incidence of reported diabetics in our society is likely to be a vast underestimation of the number of actual cases.

Diabetes mellitus is a serious metabolic disorder characterized by a defect in insulin secretion or action. Insulin is a hormone secreted by the pancreas which acts to make the body cells permeable to glucose, and glucose is the main energy source used by the body for normal metabolic processes. In cases of untreated diabetes, glucose cannot be used appropriately by the body due to insufficient insulin secretion or action. As a result, glucose accumulates in the bloodstream (hyperglycemia) and eventually spills over into the urine (glycosuria). In the absence of needed glucose, body cells metabolize fat and protein in its place. This depletes the body's energy reserves, and can lead to a build up of acidic byproducts (ketoacids). The results can be hazardous and may lead to coma or possible death.

Diabetes is readily diagnosed through the use of blood and urine tests which measure levels of blood glucose and urinary ketones. There are two major types of diabetes mellitus: Type I and Type II. Type I, or Insulin-Dependent diabetes (IDDM) usually begins early in life. It is generally considered to be the more serious, and lesser

common form of diabetes. Persons with Type I diabetes require insulin replacement in order to survive. Type II or Non-Insulin-Dependent diabetics (NIDDM) do not require insulin replacement in order to survive but may use insulin or oral hypoglycemic medication in order to achieve glycemic (blood sugar) control. Between 80% and 90% of all diabetics belong to the Type II group. It usually occurs after the age of 40, and generally does not require as rigorous control as Type I diabetes (National Diabetes Data Group, 1979). The causes of diabetes are multiple, and heredity seems to have a strong influence in its development. As there is no known cure at the present time for diabetes, it remains treatable, yet chronic.

All patients with chronic illness face periods of uncertainty, enforced role change, and repeated episodes of medical care often necessitating pain, disability, and attention to some medical regimen (Jacobson & Leibovich, 1984). However, as a chronic disease, diabetes mellitus is unique in several respects. First of all, diabetes requires diabetics to make lifelong changes in their everyday lifestyle. Diabetics must pay special attention to their individualized diet and exercise routines on a consistent and daily basis in order to avoid metabolic disruption. In addition, diabetics must take special hygienic and medical precautions in order to prevent

serious secondary complications from developing. Furthermore, they are required to learn appropriate technical skills in order to self-administer medications or insulin injections (if required), and they must be able to self-test and self-monitor their own blood glucose and urinary ketone levels. Thus, diabetes places considerable demands upon diabetics for the care, treatment, and management of their own disease condition. Second, diabetics live with the ever present threat that any laxness on their part to follow their diabetic regimen could have life-threatening consequences. Blood levels may shift rapidly and dramatically resulting in serious episodes of acidosis or hypoglycemia. Thirdly, diabetics usually live with the knowledge that strictly adhering to their diabetic regimens will not guarantee against the development of severe and debilitating secondary complications in later years. Secondary complications of diabetes include disorders such as retinopathy (ie. blindness), nephropathy (ie. kidney failure), neuropathy (ie.peripheral nerve damage), and cardiovascular disorders (ie. stroke or heart disease). Fourthly, despite all the adjustment and adaptation that is necessitated on the part of diabetics, it is adviseable that their stress levels be kept to a mimimum so as not to adversely effect their metabolic state. Fifthly, diabetes tends to be

somewhat of a "hidden" disease, and despite all the changes in lifestyle that are required on the part of diabetics and their families, they are often encouraged (and expected) to live a 'normal' existence.

As with any chronic disease, the psychological well-being of patients seems closely related to the course of the disease (Rodin, 1983). This is very true of diabetes. Knowledge and skills are only two factors that determine compliance to the diabetic self-care behavior, and subsequent metabolic status (Anderson, Nowaceck & Richards, 1989). Other likely factors include their psychological, social, and emotional well-being. These factors in turn not only influence diabetic compliance, but the disease process as well. However, relatively little is known about the diabetics' reactions to the diagnosis of diabetes and its often severe complications from the perspective of diabetics (Fisher, Jr., et al., 1982). In addition, relatively few studies have looked at the psychological well-being and adjustment of diabetics across the course of the lifespan. This study attempts to explore these areas.

Overview

This is a descriptive, exploratory study designed to assess the psychological impact that diabetes mellitus has on adult persons across the diabetic lifespan. Both Type I

and Type II diabetics are investigated in this study and comparisons are made between recently diagnosed diabetics (diabetic onset less than 1 year ago), and long term diabetics (diabetes duration 10 or more years). It is hoped that by gaining insight and understanding as to the manner in which the psychological well-being of adult diabetics is effected by the disease across the course of the lifespan, that such information will prove useful in the planning of treatment, counselling and educational programs for diabetics.

Purpose of the Study

The purpose of the study is to explore the psychological impact of diabetes on diabetics across the lifespan to see what changes may occur over time. It was anticipated that the findings of this study might reveal answers to some of the following questions:

1. How does diabetes mellitus effect the social, psychological, and physical aspects of life for diabetics both initially and after they have lived with it for several years?

2. What difference, if any, are there in the psychological functioning of persons who have just been diagnosed with diabetes in comparison with persons who have lived with diabetes for several years?

3. Do diabetics seem to adapt to their condition over time, or do adverse psychological effects of the disease increase over time?

4. What impact does the severity of the disease, secondary complications, and metabolic control have on the psychological and social functioning of diabetics?

5. What impact does physical disability have on the psychological and social well-being of diabetics?

6. Does metabolic control decline or improve across time and what relationship exists between metabolic control and psychological disorder?

7. What is the relationship between psychopathology and diabetes across time, and does the incidence of psychopathology appear to be related to metabolic control or secondary complications?

8. What similarities or differences exist between Type I and Type II diabetics with respect to psychological, social, and physical functioning?

Definition of Terms

1. Psychological: In this study, psychological refers to any covert or overt behavior of an individual. It includes all mental processes of the individual which in turn may affect behavior. Therefore, "psychological" in this study encompasses: cognition, perception, emotion, personality,

and neuropsychological or psychopathological functioning of the individual.

2. Diabetes Mellitus: a chronic, often inherited, metabolic disorder characterized by elevated plasma glucose concentration and an increased rate of gluconeogenesis (formation of glucose from non-carbohydrate substances). Symptoms include excessive thirst, increased frequency of urination, weight loss, fatigue and hunger.

3. Diabetes Mellitus Type I: Insulin-Dependent Diabetes Mellitus (IDDM). Onset of symptoms is usually abrupt, and it is generally considered to be the more severe form of diabetes. Insulin medication is required to sustain life. This type of diabetes often occurs early in life (before age 40), although it may occur at any age.

4. Diabetes Mellitus Type II: Non-Insulin Dependent Diabetes Mellitus (NIDDM). It frequently presents itself with little, if any symptoms. Generally there is a slow progression of the disease in comparison to IDDM. NIDDM usually occurs in persons over 40 years of age, although it may occur in younger persons. Insulin is not required to maintain life, but may become necessary if diabetic symptoms cannot be controlled through the use of diet or other measures.

5. Glycosylated Hemoglobin (HbA1) Assay: A method used to measure glycemic control over the previous 6 - 8 weeks

which is independent of time of day, recent food intake, or activity levels.

6. Glycemic Control: The degree to which the blood sugar (glucose) levels fall within the normal range. Also referred to in this report as "diabetic control" or "metabolic control".

7. Glycosuria: A common symptom of diabetes mellitus - elevated levels of sugar in the urine.

8. Hyperglycemia: A condition characterized by excess sugar (glucose) in the blood, usually occurring because the pancreas is not producing an adequate amount of insulin. The blood of a normal person contains between 3.6 and 6.4 millimoles/Litre of glucose after he has fasted for some hours. Two hours after a meal this glucose level may rise to as high as 7.8 millimoles/Litre in nondiabetic individuals. In untreated cases of diabetes, the fasting blood glucose level may rise above 6.4 millimoles/Litre and in some cases, glucose levels may reach levels higher than 20 millimoles/Litre two hours after a meal.

9. Hypoglycemia: A deficiency of sugar (glucose) in the blood (below 3.6 millimoles/Litre in the blood of a normal person has not eaten for some time). In diabetics, overdoseage of insulin, delay in taking food after an injection, or excessive exercise may lead to hypoglycemia. If severe enough, insulin coma may result. Swallowing of

sugar leads to rapid recovery of hypoglycemia.

10. Insulin: A hormone, produced by the Islets of Langerhans in the pancreas, which controls the metabolism of sugar (glucose).

11. Ketosis: a condition characterized by the presence of ketone bodies in the blood which produces a drop in pH that is incompatible with life. The lowering of pH is caused by the accumulation of acid byproducts (ketoacids) that result from the breakdown of fats, proteins, and carbohydrates to form glucose in the absence of insulin. This alteration in the acid-base equilibrium of the body can be critical and may lead to coma or death.

II. REVIEW OF THE LITERATURE

Rationale for Studying the Psychological Impact of Diabetes Mellitus

"How the patient feels about his diabetes - is the secret ingredient that is going to make the difference between the success or failure of therapeutic and educational programs" (Hamburg, et al., 1979).

Clinicians have long been aware of the association between subjective well-being and physical health (Connell, et al., 1988; Finkelstein & Drash, 1986), and it is now widely recognized among health professionals that psychiatric problems and social problems are common in all types of chronic medical conditions (Wilkinson, 1987). Studies have shown that the demands of following diabetic regimens can sometimes be very stressful for diabetic patients. As a result, emotional disturbance, psychiatric disorder and problems with family conflict may arise (Finkelstein & Drash, 1986; Hamburg, et al., 1979). Anxiety and depression are two of the most commonly encountered psychiatric problems of medically ill patients. In addition, depression and anxiety are often found to co-exist in persons with concomitant physical diseases, such as diabetes (Lustman, 1988). However, the psychological well-being of diabetics is crucial since much of the care and treatment for their condition is left up to themselves. Any psychological disruption that diabetics

may experience can adversely affect compliance with their diabetic regimens. This in turn can have both long and short term consequences on the course of the disease. In addition, there is evidence which suggests an association between the effects of acute or chronic stress on metabolism (Finkelstein & Drash, 1986), and between psychiatric illness and poor glucose regulation in patients with severe diabetes (Lustman, 1988). Clearly, it is important for health professionals to assist diabetics in maintaining relative well-being and functioning. This serves as a major challenge for health professionals (Holmes, 1986).

In order for health professionals to determine how to best help diabetics maintain relative well-being and functioning in our society, it is imperative that they ascertain what impact diabetes has on the life of the diabetics. Indicators of subjective well-being include not only overall physical health, but functional ability, social economic status, income, marital interaction, and social participation (Speitzer, Snyder, & Larson, 1979-1980). Also important is the degree of patient participation in ongoing follow-up care and medical treatment, continuing diabetes education, emotional support, and behavioral or coping skills training (Anderson, Nowaceck & Richards, 1989). No doubt these

influencing factors do not remain stable throughout the diabetic lifespan but will continue to influence the course of diabetes in various ways depending on changing circumstances. However, relatively little is known as to what general changes occur across the diabetic lifespan. Obviously, if diabetics are expected to live a 'normal' existence despite their illness, then it is imperative that health professionals determine what is 'normal' for diabetics. Only then can effective diabetic treatment, counselling, and educational programs be implemented.

Reactions to the Diagnosis of Diabetes and its Complications

Learning that one has a chronic disease such as diabetes mellitus can be a very traumatic experience for diabetics and their immediate family members. The development of diabetes clearly sets in motion a major life crisis that seriously challenges their adaptive capacities. (Jacobson & Leibovich, 1984). How they respond to the diagnosis and adapt to diabetes depends on several factors. Each individual will have special responses that can be understood only in light of their age, stage of development, personal history and present circumstances. Anger, anxiety, fear, depression, and denial are some of the common responses to the initial diagnosis of diabetes and to the loss of a previously perceived healthy self. In

effect, the reactions of a person who learns that he or she has diabetes may be seen as being similar to the stages suggested by Dr. Kubler-Ross for dying patients (Citrin, et al., 1986). There may be shock upon learning of the diagnosis, anger about the unfairness of having the disease, fear about the development of secondary complications or a shortened lifespan, and guilt surrounding the underlying cause of the disorder. Eventually, it is hoped that diabetics will adapt and accept their condition.

How diabetics initially respond to the diagnosis is important. Any excessive emotionality experienced by the newly diagnosed diabetic may hinder their ability or motivation to learn and perform the complex skills needed to manage their diabetes. Levine, et al., (1986), interviewed 10 NIDDM patients which were recently diagnosed with diabetes. It was found that most of the patients had a reaction of overt anxiety or depression on learning they had diabetes. Fear of death, blindness, and loss of body parts were also common. In addition, all patients reported experiencing some degree of loss of their favorite foods, of losing their freedom to unrestricted eating, and they often complained about feeling "starved" or "deprived". Similarly, Fujita, Kobayashi & Imamura (1967), in their study reported that for diabetics to receive the diagnosis

of diabetes for the first time was stressful and threatening and that it subsequently raised anxiety levels in newly diagnosed diabetics.

Psychological problems are particularly common among those who develop diabetes in early life and have not yet developed effective strategies for dealing with anger and frustration (Smith, D., 1988). In addition, clinicians have repeatedly commented that children and adolescents with IDDM seem to be more difficult to manage and that they appear to have more psychosocial problems than youngsters with other chronic medical disorders (Ryan, et. al., 1986). It is also not uncommon for individuals upon learning of their diagnosis to become hostile - blaming themselves or others for their own condition. As a result, their interpersonal, psychological, and social functioning can be seriously effected.

Diabetes diagnosed over the age of 40 or 45 years is also found to be a very traumatic experience. It tends to serve older individuals as one more direct reminder of their own mortality. Furthermore, heredity often plays a strong role in diabetes, and many of the older newly diagnosed diabetics may have knowledge of relatives who have died after the physical complications of diabetes (Holmes, D.. 1986). The personal knowledge of such events may heighten their emotional arousal, deepen their

depression, and interfere with their compliance to the diabetic regimen. Alternatively, some patients on learning of the diagnosis of diabetes may react with optimistic denial. This can have hazardous consequences as the individual who denies his or her own condition may feel that their problem can be mastered without any change in activity or lifestyle. In short, they may fail to attend to their diabetic program. However, as their diabetes becomes more difficult to manage, or its complications become more real, these overly optimistic individuals may experience delayed grief reactions (Jacobson & Leibovich, 1984). In a study of 10 NIDDM patients conducted by Levine, et al.,(1986), it was found that lack of anxiety at the time of diagnosis was often associated with noncompliance to the diabetic regimen. Of three patients who denied anxiety at the time of diagnosis, two were nonadherent to the diabetic regimen on followup. Of four patients who denied that it would be difficult to adhere to the diet, three were nonadherent to their diabetic regimen on followup.

Common reactions to the initial onset of secondary complications include: depression, anxiety, withdrawal, bodily complaints, anger, sleeplessness, guilt, shame and self-blame. In addition, diabetics may feel that they no longer have control over their own future. The results can

be devastating and ideas of suicide are not uncommon (Holmes, D., 1986). Unfortunately, suicide by noncompliance to the diabetic regimen is more common than is often thought (Calobrisi, 1983). Therefore, how diabetic individuals respond to the diagnosis of diabetes, or to the appearance of secondary complications is important. An initial response that is very negative can have serious and far-reaching consequences.

Prognosis and Secondary Complications of Diabetes Mellitus

"The diagnosis of diabetes mellitus carries with it a sinister prognosis with an overall mortality of 60% four decades after the initial diagnosis is made" (Popkin, et al., 1987). Death with, or from hypoglycemia is more common then death from ketoacidic coma, and women tend to survive longer with their diabetes than men. For those who develop diabetes before the age of 30, 50% of these patients will be dead before the age of 50. For those who live with diabetes for over 40 years, it is suspected that 16% will have gone blind, 14% will have severely impaired vision, 21% will have had a myocardial infarction, 12% will have had gangrene or have undergone amputation, 10% will have suffered from a stroke, 38% will have had proteinuria and 22% will have had uremia (Popkin, et al., 1987; Deckert, Poulsen & Larsen, 1978). Therefore,

the prognosis and outcome of diabetes can be very grim indeed. In addition, diabetes is also associated with obesity, hypertension, (Bild, et al., 1989), sexual dysfunction (Fisher, et al., 1982), and a greater use of prescription drugs in comparison to nondiabetics (Isacson & Stalhammer, 1987). According to the results from Isacson & Stalhammer's study (1987), diabetics were found to receive a greater number of prescriptions for cardiovascular drugs, diuretics, central nervous system drugs and psychotropics, antibiotics and chemotherapeutics, analgesics, respiratory system drugs, dermatologicals and hormonal drugs, in comparison to the nondiabetic population. Also, senile cataracts appear at a earlier age in diabetics compared to nondiabetics. The presence of senile cataracts in diabetics necessitates surgical excision with five times the frequency of nondiabetic individuals (Clements & Bell, 1985).

Hyperglycemia is believed to be one of the major contributers to the development of diabetic complications. However, strict adherence to the diabetic regimen does not necessarily prevent secondary complications from developing. Other likely influences include genetic predisposition, hypertension, and environmental factors (Godine, 1988).

i) Retinopathy and Diabetes

Blindness is 25 times more common in diabetic patients than in nondiabetics, and is due to proliferative diabetic retinopathy (PDR) (Clements & Bell, 1985). It is the commonest cause of blindness in persons between the ages of 30 and 64 (Owens, et al., 1988), and it's development is believed to be related to poor metabolic control and diabetic duration. For patients with less than a 10 year duration of diabetes, the rates of proliferative retinopathy are 4.9% for insulin users, and 2.6% for nonusers. For patients with greater than a 10 year duration of diabetes, the rates of proliferative retinopathy are 17.7% for insulin users and 3.9% for nonusers (Klein, Moss & Klein, 1987). While the prevalence of diabetic retinopathy is much greater in the Type I group, the NIDDM patients constitutes the largest population of retinopaths. This is because the majority of persons with diabetes are Type II diabetics. Up to 60% of all cases of diabetic proliferative retinopathy are NIDDM individuals, and approximately 20% of NIDDM patients already have retinopathy when they are first seen at the diabetic clinic (Owens, et al., 1988). Diabetic retinopathy is not usually observed in patients with Type I diabetes until the disease has been present for at least 5 years. Thereafter the prevalence of retinopathy increases

dramatically to 80% after 30 years of diabetes (Clements & Bell, 1985).

Proliferative retinopathy often leads to at least partial visual impairment, psychiatric symptoms and difficulties with glycemic control. Furthermore, fluctuating visual acuity is common in proliferative retinopathy which often leads to feelings of uncertainty, fear and hope (Wulsin, Jacobson & Rand, 1987). One source of disruption for some patients is the restriction of physical activity which visual impairment causes. These limitations interfere with choices of jobs, domestic duties, outside activities, and lifestyle in general. In addition, diabetic retinopaths are often faced with the realization that they must become more dependent on others. Consequently, living with diabetic retinopathy can be quite distressing. One cross sectional study (Oehler, 1978), investigated the psychosocial impact of diabetic retinopathy and recent visual loss in 33 adults. His study revealed that 33% of the subjects reported "severe depression", 69% reported work difficulty related to vision, 39% reported losing their jobs, 73% could no longer drive, and 39% began receiving support from social security (Wulsin, Jacobson & Rand, 1987). Similarly, Jacobson & Leibovich, (1985), compared 74 diabetic patients who had

various durations of proliferative retinopathy with 76 patients who had a long duration of diabetes with no or minimal retinopathy. They found that there was a trend toward an increase in negatively experienced life events during the first two years after the onset of proliferative retinopathy regardless of the degree of visual impairment. In addition, they found a higher frequency of psychiatric symptoms in patients with both recent and long standing proliferative retinopathy. In summary, the onset of diabetic retinopathy or visual impairment calls for major adjustment in the lifestyle of diabetics. This can have a profoundly negative impact upon their psychological well-being.

ii) Peripheral Vascular Disease and Diabetes

Peripheral Vascular Disease (PVD) can cause poor healing of foot lesions, ulcers, and gangrene leading to amputation. It generally involves vessels below the knee and is more often found in diabetics than nondiabetics. Peripheral vascular disease eventually affects about 30% of diabetics (Holmes, 1986). Prevalence increases with age, duration of diabetes, and is more commonly found in men than women. Those with IDDM have been found to have a slightly higher prevalence of PVD than those with NIDDM after adjusting for age (Bild, et al., 1989).

iii) Lower Extremity Amputation

Individuals with diabetes experience a rate of lower extremity amputation (LEA) about 15 to 16 times that of people without diabetes (Bild, et al., 1989; Holmes, D., 1986). Furthermore, the three year survival rate for people with diabetes who have undergone an LEA is only about 50%. It is believed that diabetic individuals are predisposed to LEA primarily as a result of the presence of peripheral neuropathy and peripheral vascular disease. However, other factors also contribute. It has been estimated that more than 50% of the amputations which occur in the diabetic population could be avoided by improving foot care, hygiene, and metabolic control (Bild, et al., 1989). Generally, the incidence of LEA increases correspondingly with diabetic age and duration. Approximately 64% of all LEA occur in those aged 65 or older and 96% in those aged 45 years or older. Because the risk of amputation increases with age, and the NIDDM is older and more than 10 times the IDDM population, the overwhelming majority of amputations occur in individuals with NIDDM (Bild, et al., 1989).

Both patients with limb amputation and those persons closest to these patients are deeply and personally affected by the amputation. The postamputation period calls for major adjustment in lifestyle. However, the first reaction of the diabetic to the LEA is often one of

relief followed by sadness. Loss of appetite, sleep difficulties, self-blame, despair, and phantom limb pains also frequently accompany the postsurgical period. How well diabetics adjust to LEA depends on their physical and psychological characteristics and the physical and psychological characteristics of their families (Holmes, D., 1986).

iv) Nephropathy and Diabetes

Small blood vessels affecting the kidney and eventually leading to renal failure, occurs at least 17 times more often in diabetics than nondiabetics (Clements & Bell, 1985; Holmes, D., 1986). It is particularly common in Type I diabetics, but less so in Type II diabetics. Approximately 40% of patients with Type II diabetes never develop nephropathy regardless of the degree of glucose control (Godine, 1988). However, of patients in which diabetes develops before the age of 20 years, roughly 70% will have proteinuria after 20 years of diabetes (Clements & Bell, 1985). The outcome of nephropathy can be fatal as renal failure ultimately leads to death. Therefore, diabetics confronted with the serious symptoms of nephropathy are likely to experience severe psychological disruption, and may undergo loss and grief reactions similar to those described by Dr. Kubler-Ross.

v) Neuropathy and Diabetes

At the onset of diabetes, most signs and symptoms of neuropathy are usually not apparent except for abnormal nerve conduction velocities which are present in virtually all diabetics. Neuropathic signs and symptoms do, however, become progressively worse with increasing diabetic duration. Signs of peripheral nerve dysfunction include numbness, reduced pain and thermal sensation, abnormal pain and light touch sensation, and painful paresthesias. In patients with good metabolic control, these symptoms are present in about 10% of all diabetics, and in persons who have had diabetes for 30 years or more, this may reach 50%. In diabetics with a history of poor glycemic control, the prevalence of neuropathic signs and symptoms can reach almost 70% after 25 years of diabetes (Bild, et al., 1989; Clements & Bell, 1985). The appearance and discomfort of advancing neuropathy may produce psychological distress for its victims, including feelings of self-blame, depression, anxiety, and fear.

vi) Sexual Dysfunction and Diabetes Mellitus

Impotence and disturbances in sexual functioning are frequent complications of diabetes. In males, diabetes is the single most common cause of impotence. It is commonly noted within 10 years after diabetic onset and is found with 2 - 5 times the frequency in diabetics in comparison

to nondiabetics (Whitehead, 1987; Whitehead, et al., 1983; Schmitt, Stein & Neubeck, 1985). Research findings suggest that the longer the duration of diabetes, the greater the probability of sexual dysfunction (Schmitt, Stein & Neubeck, 1985). Approximately 15% of diabetic men between the ages of 30 and 40 are impotent, and about 55% of diabetic men are impotent by age 60 (Whitehead, 1987). However, sexual dysfunction is not limited to male diabetics, as female diabetics also report experiencing sexual difficulties. In a study of 125 diabetic women, and 175 diabetic men, Fisher, et al., (1983), found that 35% of the diabetic women reported orgasmic dysfunction in comparison to 6% of nondiabetic controls. Of the diabetic men, 49% reported impotence, 2% reported premature ejaculation, and 1% reported retrograde ejaculation. Similarly, Schmitt, Stein & Neubeck (1985), in their study found that of 77 diabetic males, 64% reported experiencing some degree of impotence or erectile difficulty and 71% of these men reported experiencing sexual dysfunction of the 'severe' category. 82% of these men were over the age of 50.

Difficulties with sexual functioning can seriously harm the communication and relationship between couples. However, it has been found that when couples have knowledge of the physiological linkage between diabetes and sexual

dysfunction, that they are better able to adjust to the effects of diabetes on their sexual and marital relationship. When couples are not aware of this linkage, serious marital conflict can result as both partners may exhibit behaviors destructive to the marriage (Schmitt, Stein & Neubeck, 1985). These findings attest to the importance of diabetic knowledge and education in improving life for diabetics and their families.

Adherence or Non-Adherence to the Diabetic Regimen

According to the Becker and Maiman Health Belief Model, the likelihood of an individual taking action to adhere to a medical regimen is determined by five psychological readiness factors. These include: perceived suseptibility to the disease process, perceived severity of the condition, perceived benefits of taking action, perceived costs of taking action, and the degree to which internal or external cues to action are present to activate the other psychological readiness variables. Researchers have found that compliance is also effected by perceived blood-glucose changes, beliefs about symptom-blood glucose relationships, (Diamond, Massey & Covey, 1989), locus of control orientation (Alogna, 1980), and knowledge about the disease process (Delbridge, 1975). In a study of 143

insulin dependent diabetics (diabetic duration at least 1 year), it was found that perceived benefits and perceived severity were associated with greater self-reported adherence to the diabetic regime and good metabolic control, while perceived costs were associated with poorer metabolic control and self-reported adherence to the diabetic regime. Greater perceived suseptibility to complications was associated with poorer metabolic contol, and in older diabetic patients (longer diabetic duration), adherence to the diabetic regimen was predicted by perceived benefits. For the younger diabetic patients (shorter diabetic duration), adherence to the diabetic regimen was predicted by severity and susceptibility to complications (Brownlee-Duffeck, et al., 1987). This suggests that patients who have had diabetes for a longer duration may be more likely to take a preventative approach to treating their diabetes, while younger diabetics may be more concerned about the immediate effects of compliance.

Stress, Stress Hormones and their Effects on Diabetic Control

Psychological factors have a powerful influence on diabetic metabolism through the mechanisms of the sympathetic nervous system. The sympathetic nervous system controls the release of anti-insulin hormones, thereby effecting blood glucose levels (Bradley, 1982). Emotional

arousal, such as fear or anxiety stimulates the sympathetic nervous system and causes the pituitary and adrenal glands to release stress hormones. These hormones regulate the metabolism of carbohydrates, lipids, and proteins, and the secretion of insulin from the pancreatic islets. Stress hormones such as epinephrine, norepinephrine, glucagon, growth hormone, and cortisol are released when an individual undergoes emotional stress. Among the many biological effects of these hormones, they tend to produce a rise in blood glucose by counteracting the effects of insulin. Emotional stress can lead to disruption of metabolism in the diabetic individuals who is physiologically unable to compensate for a rapid reduction in insulin or the sudden elevation of blood glucose.

In the 1950's, Hinkle and Wolf, conducted one of the first experiments which attempted to determine the association between stress and diabetic control. In their experiment, 64 diabetic patients underwent stressful interviews while in the fasting state. They were not given any insulin for 24 hours prior to the interviews. Results from this experiment showed increased urine volume, increased blood glucose, and increased ketone levels. Metabolic changes observed tended to be more extreme in the severe diabetic cases. Similar studies have produced contradictory findings. Vandenbergh and his co-workers,

(1967), used hypnotically induced emotion and anticipation of electric shocks in diabetics to determine whether emotional stress might influence diabetic control. Contrary to Hinkle and Wolf's results, they found that emotional arousal tended to reduce blood glucose levels. Another study investigating the association between examination stress in University students and blood glucose levels found that stress decreased blood glucose levels prior to and during exam week (Vandenbergh et al., 1967). Therefore, it seems that stressful situations sometimes cause an increase, and sometimes cause a decrease in blood glucose. Why this is so is not clearly understood.

In order to evaluate the mechanism of rebound and stress hyperglycemia in diabetics, Shamoon, Hendler & Sherwin, (1980), infused cortisol, epinephrine and glucagon, into diabetics and nondiabetic subjects for 5-6 hours in doses designed to simulate the metabolic changes observed in insulin hypoglycemia or severe stress. Results showed that the infusion of cortisol and epinephrine produced similar elevations in plasma cortisol and plasma epinephrine in both groups of subjects. However, the increment in plasma glucose in diabetics after cortisol and epinephrine infusion was 5-7 times greater than that in normals. Glucagon infusion produced a 35-40% rise glucose production in both nondiabetic and diabetic groups.

Therefore, results from this study suggest that diabetics have increased sensitivity to anti-insulin hormones such as epinephrine and cortisol. Consequently any changes in the release of these hormones as a result of emotional stress, has the potential to significantly alter metabolic control in diabetics.

Metabolic Control and Mood Changes

Metabolic imbalance is believed to account for powerful and unpredictable changes in mood that are sometimes seen among diabetics (Hamburg, et al., 1979). Studies have found that abnormally high or low blood glucose levels often correlate with changes in self-perceived mood in diabetics. With low blood glucose levels, diabetics tend to report experiencing anxiety and irritability. With high blood glucose, diabetics tend to report they feel themselves in a better mood. Subjectively they have used terms such as "sharp" or "on top of things" to describe their state while experiencing hyperglycemia (Gonder-Frederick et al., 1989). When 52 Type II diabetics were asked "Can you tell just by how you feel when your blood sugar is too high, 77% responded "yes". When the same subjects were asked "Can you tell when your blood sugar is too low, 40% responded "yes". Furthermore, 35% of the subjects claimed that they could detect both high and

low blood sugar. High blood glucose was subjectively reported as producing somatic complaints of dizziness, light-headedness, thirst, frequent voiding, weakness, and blurred vision. Low blood glucose level was subjectively detected by reported feelings of hunger, nausea, feeling faint, and dizziness. Dry mouth, thirst and frequent voiding were associated with the experience of both high and low blood sugar levels. According to the results of this study, diabetics are usually quite accurate in estimating their own blood glucose levels. Numerical guesses of their current blood glucose level were often found to correlate with their actual blood glucose levels. However, diabetics tended to underestimate their current blood glucose level if their glucose level was especially high (Diamond, Massey & Covey, 1989). The results from these studies indicate that diabetics are not only aware of their own subjective feelings and mood states, but they are able to use this information to predict their own degree of metabolic control quite well.

The Association between Metabolic Control and Psychological Well-Being

Recent studies have provided substantial evidence which suggests that persons in poor metabolic control may take a more negative view of themselves, a more

negative view about life, and that they may have greater difficulty with social functioning. In a study conducted by Orr, et al., (1983), it was found that diabetics in poor control had poorly developed social skills, reported few or no close friends, did not date, and avoided parties due to shyness and an inability to converse with peers. Similarly, results of a study utilizing Eysenck's Personality Inventory, found diabetics in poor control to be more introverted with lower sociability and curiosity scores (Lane, et al., 1988). Schaefer, et al., (1983) found IDDM children in poor glycemic control to be more hypoactive in comparison to diabetics in better control. Diabetes is also often associated with lowered self esteem (Jensen, 1985), and a poorer self concept in comparison to nondiabetics (Ryan & Morrow, 1986). These features tend to be more apparent when metabolic control is poor. Diabetics in poor metabolic control also seem to undergo greater metabolic disruption during stressful events, and then may find their diabetes much more difficult to cope with (Delamater, et al., 1987). Feelings of low self esteem, anger and guilt directed towards self, and poor sociability skills, are likely hinder the functioning of the diabetic individual in everyday life. Their negative feelings will likely serve as a source for further problems and conflicts in life.

Both a high internal locus of control and a high external locus of control have been associated with poor metabolic control. Persons with a high internal locus of control may have a tendency to blame themselves for their poor metabolic state. (Peyrot & McMurry, Jr., 1985). Self blame may contribute to feelings of low self worth and depression in the individual. Alternatively, persons who tend to have a high external locus of control may blame others for their poor metabolic state. Consequently, they may look to others to remedy their problem. In addition, they may abdicate any attempts to control their own blood glucose levels. Therefore, individuals with either a very high internal or external locus of control may be at a higher risk for demonstrating symptoms of poor glycemic control.

Adjustment and Adaptation in Diabetes Mellitus

Popkin and his colleagues (1987), found that Type I diabetics were characterized by increased rates of divorce and separation, unemployment, and multiple hospitalizations in comparison to a group of 1002 patients with other medical illnesses. Their findings point to the disruptive impact that diabetes can have on the lives of individuals with the disease.

It appears that how well diabetics adapt to their diabetes will influence their metabolic control, and this in turn may effect other areas of their life (Felton,

Revenson & Hinrichsen, 1984). Furthermore, how well diabetics adapt to their disease is determined at least partially by how effective their coping skills are. What coping skills are utilized will depend on how they perceive stressful situations. It has been found that patients in poor metabolic control utilize different coping strategies than do those patients in good metabolic control. Persons in poor control have been found to utilize inefficient coping strategies, such as wishful thinking, (wishing the situation would change or hoping for a miracle), and avoidance/help seeking behavior (refusing to believe it happened, avoiding being with people, and getting away from it for awhile). Patients in poor control also tend to utilize what is known as a "scatter shot" approach to coping moreso than do those in good metabolic control. With the scatter shot approach, the individual attempts to utilize a variety of coping strategies in the hopes of findings one that may work best for them (Delamater, et al., 1987). Alternatively, patients in good metabolic control tend to use more information seeking (Felton, Revenson & Hinrichsen, 1984), and educational type coping methods. That is, they attempt to learn as much as they can about their own condition in order to deal more effectively with it. Appropriate self-care has also been associated with better metabolic control (Kaplan, Chadwick

& Schimmel, 1985). Once a diabetic develops good habits of self-care and self-management a certain level of metabolic control is achieved, these habits and patterns have been shown to remain relatively stable across time. In a study assessing the effects of self monitoring of blood glucose on long term glycemic control (IDDM) patients, it was found that the patients' control worsened during the first year after diagnosis; it improved slightly during the second year, but there was no statistically significant change over an entire 3 year period (Belmonte, et al., 1988). It therefore appears that the year after diabetes calls for major changes in lifestyle. This requires patients to learn their personal diabetic routines, and learn the necessary skills for self-management. Once these knowledge and skills have been acquired and practiced, diabetic control improves somewhat and remains relatively stable across time.

After the emotional upheaval induced by the initial diagnosis of diabetes, the appearance of secondary complications once again is a traumatic time demanding major adaptation and adjustment in lifestyle. Literature on chronic illness suggests that those who adapt most readily to complications have adequate income and housing, sustaining relationships and membership in support groups, affection and the respect of others, congeniality, freedom

from oversensitivity, a vital interest in life, tolerance, and a willingness to shoulder responsibility (Holmes, D., 1986). Presumably, the individual's ability to adapt to having a chronic illness such as diabetes, will predict to a certain extent, how well they will adapt when secondary complications arise.

Cognitive and Neuropsychological Changes Associated with Diabetes

Research conducted during the past 20 years has provided strongly suggestive evidence that diabetes (especially Type I diabetes) affects the integrity of the central nervous system and can produce serious cognitive and neuropsychological deficits (Ryan, et al., 1984; Rovet, Ehrlich, & Hoppe, 1988; Ryan & Morrow, 1986). Several studies performed by various researchers have found significant impairment in some diabetics for tasks requiring visuospatial, verbal, mathematical, motor strength and motor speed skills. Also, decrements have been found in diabetics for tasks requiring good attentional, somatosensory discrimination and visual-motor coordination skills (Holmes, C., 1986; Ryan, et al., 1984; Rovet, Ehrlich & Hoppe, 1988; Ryan & Morrow, 1986; and Skenazy & Bigler, 1984). Memory seems to be little affected in most diabetics (Skenazy & Bigler, 1984). Furthermore, encephalographic (EEG) recordings often reveal

irregularities in brain wave patterns of many diabetics (Ryan, et al., 1984). In 1966, Eeg-Olofsson reported abnormal EEG patterns in 35% of diabetic children he assessed between the ages of 2-15. It is has been hypothesized that damage to the central nervous system (CNS) is produced by prolonged and severe hypoglycemia. The effects of this damage are believed to be responsible for this "diabetic encephalopathy" (Holmes,C., 1986; Ryan, et al., 1984). Decrements in cognitive and intellectual functioning appear to be most common in those individuals who develop diabetes very early in life (particularly before age 3 and 4), and/or have had diabetes for a long duration; in those who have demonstrated poor metabolic control and/or in those who have undergone severe hypoglycemic episodes (convulsions or coma) (Holmes, C., 1986; Jacobson & Leibovich, 1984; Ryan et al., 1984; Rovet, Ehrlich & Hoppe, 1988; and Ryan & Morrow, 1986).

Experimental neuropathological studies have shown that hypoglycemia can cause lesions in the brain which are similar to those which are caused by hypoxic insults. Microscopic lesions have been found in the cerebral cortex and the hippocampi of diabetic individuals. Also, lesions in the intraparenchymal cerebral vessels have been found to be 25 times more common in diabetics than in nondiabetics. Post mortem examinations have shown that in the age range

30-70 years, nearly twice as many diabetic as nondiabetic persons have moderate or severe cerebral atherosclerosis (Wilkinson et al., 1981). It would appear, therefore, that there is substantial evidence which suggests that hypoglycemia may have an adverse effect on the physical, and neuropsychological functioning of the diabetic individual.

Personality, Psychiatric Disorders and Psychopathology in Diabetics

(i) Is there a Diabetic Personality?

For many years it was felt that a specific diabetic personality existed (Finkelstein & Drash, 1986). Going back as far as 1679, Thomas Willis hypothesized that prolonged sorrow was important in the etiology of diabetes. In 1899, Henry Maudsley claimed that diabetes was a disease that often became apparent in families in which insanity prevailed. 250 years after Willis' speculation, Menninger, came forward with his belief that emotional stress, anxiety, and depression were features frequently found in the diabetic personality (Dunn & Turtle, 1981; Tattersall, 1981). According to Menninger, characteristics of the diabetic personality included: diminished alertness, apathy, hypochondriacal concerns and vulnerability to depression. Part of the basis on which he arrived at these conclusions arose from experiments which were conducted by

Canon in 1911. These experiments demonstrated that glycosuria was part of a normal "stress reaction" which could be induced by fright or rage (Tattersall, 1981). In addition, many of the early theories were also heavily influenced by the psychoanalytic movement. According to early psychoanalytic theory, only certain personality types could develop certain psychosomatic diseases. Following this line of theory, Dunbar in 1943, claimed that the personality characteristics which predisposed one to diabetes included: weakness, irritability, hypochrondriasis, frequent mood swings, and frequent changes in behavior ranging from overdependency to rebelliousness (Lloyd, Steel & Young, 1987; Tattersall, 1981). Later on, the psychoanalytic movement took a different approach to psychosomatic diseases after it was claimed that unconscious conflicts were responsible for the development of specific psychosomatic diseases (Alexander, 1950).

Despite much theory and research attempting to determine the characteristics of the diabetic personality, results have consistently failed to support the notion of a particular personality pattern common to all adult diabetics. In addition, no relationship has been found between glycemic control and personality (Mazze, Lucido, and Shannoon, 1984). Therefore present day theorists contend

that no one diabetic personality type exists (Surwit, Feinglos & Scovern, 1983).

ii) The Prevalence of Psychiatric/Psychological Disorders in Diabetics

Recent studies have indicated that the prevalence of psychiatric disorders is higher in diabetic patients in comparison to nondiabetics. In 1986, Lustman found in his study of 144 diabetic patients, that 71% of them had a history of at least one defined psychiatric illness. Affective and anxiety disorders were the most common diagnosis with 33% experiencing a major depressive disorder, and 41% of them reporting generalized anxiety disorder. Agoraphobia and simple phobia occurred significantly more often in Type II diabetics when compared to Type I diabetic. However, no significant difference was found between Type I and Type II diabetics with regards to the overall prevalence of psychiatric illness (Lustman, 1986). Another study conducted by Lustman, Griffith and & Clouse, (1988), found that out of 114 adult diabetics, 32.5% had a major depressive disorder at some point during their life, 38.6% had other psychiatric illnesses, and 28.9% had no history of psychiatric illness. In addition, 37 recently diagnosed patients were followed up after a five year period, and it was found that 64% of them had experienced at least one episode of depression since

initial diagnosis. According to results from their studies, more than half of the diabetic population experiences some form of psychiatric illness. Similar results were obtained from a study conducted by Popkin and his colleagues in 1988. In their study of 75 persons with long duration type I diabetes (diabetic duration at least 10 years), 51% of them received one or more psychiatric diagnosis in comparison to 33% of the general population (after tobacco use disorder and psychosexual dysfunction were excluded from analysis). Lifetime prevalence rates for major depression, generalized anxiety disorder, phobic disorder and psychosexual dysfunction ranged from 24-32%. Major depression was found to have a lifetime prevalence of 24% while generalized anxiety disorder was found to have a lifetime prevalence of 31.7%. In addition, females were found to have a higher lifetime prevalence of simple phobia, whereas males were found to have a higher lifetime prevalence of antisocial personality disorders in comparison to the general population. Swift, Seidman & Stein (1967), used psychiatric interview, psychological testing, and sociological assessments to evaluate 50 IDDM patients and 50 matched controls. Results indicated that diabetics had more psychiatric illness, more problems with dependence and independence, poorer self-perceptions, greater anxiety, greater constriction,

more pathological hostility, and poorer relationships at home and with peers in comparison to nondiabetics. Therefore, the above noted studies provide considerable support for the contention that there is a strong association between psychiatric disorder and diabetes, at least in many diabetics. However, one of the difficulties with studies of this nature is arriving at accurate psychiatric diagnosis. Identification and classification of psychiatric disorders can often be influenced by the subjective nature involved in making diagnosis. As there is no laboratory test nor definitive objective data which clearly identifies a specific psychopathology, differences of opinion regarding diagnosis frequently arises between medical professionals. In addition, professionals may use different criteria in order to arrive at diagnosis. In a study of 211 IDDM patients attending an outpatient clinic, a two-stage psychiatric screening procedure found that physicians identified 10% of the patients as exhibiting psychiatric morbidity. In comparison, psychiatrists using the Clinical Diagnostic Interview Schedule found that physicians correctly identified only 28% of the psychiatric disorders. According to the psychiatrists, the estimated prevalence of psychiatric morbidity among the 211 IDDM patients was 18% (Wilkinson, et al., 1987).

iii) Depression and Anxiety in Diabetes

Perhaps the most consistent finding in research investigating the link between psychiatric disorder and diabetes, has been the high prevalence of depression and anxiety disorders found in diabetic individuals. Several studies conducted have shown that diabetics tend to score higher on the anxiety and depression scales of the Minnesota Multiphasic Personality Inventory (MMPI). Murawski, et al., (1970), undertook a longitudinal study of 112 long duration diabetics (diabetic duration at least 25 years). In this study, subjects were asked to complete the first 399 items of the MMPI prior to scheduled physical examination. Results from their study showed that chronic diabetics scored higher on the Depression scales than the general population. This was regardless of sex or degree of vascular complications. Other studies report similar findings. In Skenazy & Bigler's 1985 study, an abbreviated form of the MMPI (Faschingbrauer Abbreviated MMPI - FAM), was administered to two groups of individuals - diabetics and nondiabetics who had other chronic illnesses. Results showed that both groups of individuals had significant elevations on the first three clinical subscales of the FAM: Hypochrondriasis, Depression, and Hysteria. This triad is often referred to as the "neurotic triad" among professionals. Individuals who demonstrate this pattern on

the MMPI and abbreviated forms of the MMPI, are typically considered to be depressed, dissatisfied, anxious, and possibly, overly concerned with regard to their health. However, overconcern with physical health or symptamatology may be considered normal in individuals with chronic medical disorders. That is, these patients may be viewed as having legitimate reasons for having a number of somatic complaints and concerns. It appears that all persons experiencing a chronic illness may be predisposed to many psychological disorders including depression, excessive anxiety, and a neurotic tendency or preoccupation with somatic symptoms.

iv) The Association between Metabolic Control and Psychiatric or Psychological Disorder.

Recent research has confirmed an association between depression and hyperglycemia in diabetics (Rodin, 1983). This association has often been explained by poor adherence or compliance with the diabetic regimen, particularly among those diabetics who are psychiatrically disturbed (Fonagy, et al.,1987). In general, it has been found that diabetics who have good mental health are usually in better metabolic control than diabetics who are in poor metabolic control. It is possible that patients suffering from depression may experience symptoms which make it difficult to follow their daily regimen. On the other hand, the experience of

hyperglycemia may produce physiological changes that predispose the individual to depression. Diabetes has also been found more often in manic-depressive patients than in the general population (Tsuang, Perkins & Simpson, 1983). Lilliker (1980), identified diabetes in almost 10% of a sample of 203 manic depressives (diagnosed according to the DSM II), while the general population rate was only 2%.

Researchers have theorized that poor metabolic control may actually produce damage to the central nervous system, and that these damages may produce profound psychiatric changes. A descriptive study of 50 IDDM patients found that 31 of them complained of increased fatigue and energy loss. 1/4 of them reported feeling "blue" or "down in the dumps", and of those who reported these symptoms, most could not understand nor account for their feelings. 18/50 reported symptoms of irritability and 8/50 reported difficulties with short term memory and concentration (Surridge, et al., 1984). Whether caused by central nervous system damage, or poor adherence to the diabetic regimen, it appears that diabetics are more likely to experience symptoms of depression and that these symptoms often correlate with blood sugar levels. Mazze, Lucido & Shamoon, (1984), studied 84 IDDM patients, and found that there was a significant relationship between changes in HbA1 levels (glycemic control) and changes in levels of

depression. According to the results of their study, as metabolic control worsened, depression scores tended to increase. Correspondingly, as metabolic control improved, depression scores tended to decline. Furthermore their study also found a significant correlation between anxiety, quality of life and metabolic control. Lower anxiety, and better quality of life were recorded for those diabetics in good control (HbA1 <8.9%) when compared with those in average control (HbA1 9.0-11.9%), and those in poor control (HbA1 >11.9%). It therefore appears that psychological well-being is strongly related to the degree of metabolic control in diabetics.

SUMMARY OF LITERATURE REVIEW

According to research, diabetes mellitus is a serious, chronic, metabolic disorder which has the potential to effect and virtually disrupt every aspect of life for diabetics. As a disorder of metabolism, diabetes can alter numerous physiological, psychological and cognitive processes. It may even produce damage to the central nervous system causing profound psychiatric and psychological changes. How well diabetics react to the diagnosis, to the onset of secondary complications and to unwelcome changes in lifestyle is crucial. A very negative attitude and future outlook contribute to further metabolic and psychological disruption. In addition, diabetics who

take on a pessimistic attitude, or who have difficulty with accepting their condition, may be more likely to develop serious physical symptoms and complications. Possibly, it may even speed up the process. How well diabetics adapt to their chronic disease and all that it encompasses, is largely determined by the perceived severity of their disease and how effective their coping skills are.
In addition, how diabetics perceive their condition will largely determine how much responsibility they will take upon themselves in controlling their own condition. As a lifelong chronic disease, diabetes calls for diabetics to undergo repeated episodes of adjustment, re-adjustment, and adaptation. However, relatively little is known about how these repeated cycles of change may effect the psychological processes of diabetics. Furthermore, very few studies have looked at diabetes and it's effects on life from the viewpoint of diabetics. No doubt the profound, and sometimes traumatic changes which occur across the diabetics lifespan, will produce changes not only in physical functioning, but in psychological and social functioning as well. Therefore, this study attempts to explore the experience of diabetes from the viewpoint of diabetics, with a focus on differences between short duration and long duration diabetics.

III. METHOD

Subjects

The subjects for this study consisted of diabetic persons attending the diabetic and metabolic centre at the Edmonton General Hospital between July 21 and August 29 and on Tuesdays between September 6 and November 15, 1988. Subjects were categorized on the basis of type, and duration of diabetes into four groups: Type I - recently diagnosed (diagnosed less than one year prior), Type I - long term (diagnosed more than 10 years prior), Type II - recently diagnosed (diagnosed less than one year prior), and Type II - long term (diagnosed more than 10 years prior). Patients who had been diagnosed with diabetes less than one month prior were excluded from the study because it was felt by the diabetologist that some of the questionnaire items might cause them undue stress. In addition, three other diabetic patients were excluded from the study because the nurses felt that they were too disturbed to respond to the questions appropriately. Lastly, nine patients were eliminated from the study, because they were not proficient in the English language. In all, 154 patients were asked to participate. Of these, 136 agreed, giving a response rate of 88%. Seven of the patients fell into the Type I-recent group, thirty-seven patients fell into the Type I - long term group,

thirty-four patients fell into the Type II - recent group, and fifty-eight of the patients fell into the Type II - long term group.

Procedure

Patients attending the clinic were asked to participate in a research project that was looking at the impact of diabetes on the life of diabetics. Subjects were provided with information regarding the type and form of questionnaires, the types of questions that would be asked, and the approximate length of time it would take them to complete the questionnaires. Those subjects agreeing to participate were asked to sign a written consent form confirming their agreement. Verbal instructions were given as to the procedure for completing the questionnaires. Written instructions were also provided within the questionnaires and proceded each set of questions. One individual administered the questionnaires in the same order to all participants. The time required to complete the questionnaires varied from thirty-five minutes to two and one-half hours. Subjects were allowed to either complete the questionnaire in the clinic, or could take the questionnaire home to be completed and returned at a later date. Subjects who chose to complete the questionnaire in the clinic were allowed to ask the person administering the questionnaires any questions they might have.

Information regarding the subjects' age, sex, employment, diagnosis, date of diagnosis, treatment, glycosylated hemoglobin level, obesity, and various secondary complications were copied from the patient's medical charts which were kept in the clinic.

Study Design

This was an exploratory study which utilized demographic data and pencil and paper type questionnaires in order to investigate the psychological impact that diabetes has on the life of diabetic individuals. In addition, the information gathered was categorized and compared according to diabetic type and diabetic duration to attempt to determine what changes might occur across the lifespan of the diabetic, and to see what similarities or differences exist between the diabetics with respect to the many physical and psychological variables.

The following questionnaires were given:

1. Campbell's Scale of Wellbeing: is a scale which has often been used in medical research. It was originally designed to assess the quality of life of the American public and norms are based on a probability sample of 2,164 Americans. Reliability of this scale is very good (Cronbach's alpha = .89). The Campbell's Scale of Well-Being consists of three submeasures: the Index of General Affect, the Overall Life Satisfaction

Index, and the Index of General Well-Being. The Index of General Affect consists of eight opposing items: boring vs. interesting, miserable vs. enjoyable, useless vs. worthwhile, lonely vs. friendly, empty vs. full, discouraging vs. hopeful, disappointing vs. rewarding, and "doesn't give me a chance" vs. "brings out the best in me". Subjects are asked to indicate where on a 7 point Likert scale they believe they fall with respect to the 8 items. The Overall Life Satisfaction Index consists of one single question in which subjects are asked to determine how satified they feel they are with their life as a whole. A 7 point Likert scale is also used with the Overall Life Satisfaction Index. The Index of General Well-being is calculated from a composite of the scores obtained on the Overall Life Satisfaction Index and the General Affect Index.

2. The Sickness Impact Profile (SIP): is a scale that is designed to assess the functioning of physically ill persons. This assessment instrument is composed of twelve subscales: ambulation (12 items), mobility (10 items), body care and movement (23 items), emotional behavior (9 items), alertness behavior (10 items), social interaction (20 items), communication (9 items), sleep and rest (7 items), eating (9 items), home management (10 items), work (9 items) and recreation and pasttimes

(8 items). In each subscale, subjects are asked to check off those items which they feel describes them on that particular day as a consequence of their state of health (in this case, diabetes). For example, on the emotional behavior scale subjects might respond to the "I talk about the future in a hopeless way" by placing a check mark beside that item if they felt it applied to them. Completion of this scale yields three total area scores: i) A physical dimension score, which is obtained from a combination of the ambulation, mobility, and body care and movement subscores. ii) A psychosocial dimension score, which is obtained from a combination of the social interaction, communication, alertness behavior, and emotional behavior scores. iii) A total sickness impact score (SIP) which is obtained from a combination of all twelve subarea scores.

Internal consistency (r=.94) and coefficient of stability (r=.92) are very high for the Sickness Impact Profile. It has also been found to correlate with self-assessments and clinical assessments of dysfunction: (r=.52), and (r=.49), respectively.

3. Jackson's Basic Personality Inventory (JBPI or BPI): is a twelve scale, 240 item true-false questionnaire designed to measure both personality and psychopathology. Subjects are asked to respond to

statements which describe activities, interests, and behavior. The twelve scales of the BPI include: Hypochrondriasis, Depression, Denial, Interpersonal Problems, Alienation, Persecutory Ideation, Anxiety, Thinking Disorder, Impulse Expression, Social Introversion, Self-Depreciation, and Deviation. Each subject receives a score on each scale which is then compared to the norming sample. Adult norms for the BPI were based on a random sample of 1237 Americans and 182 Canadians. Internal consistency coefficients range from .69 to .86 and test-retest reliabilities range from .64 to .85. The constructs of this instrument are based on a canonical factor analysis of the Minnesota Multiphasic Personality Inventory and the Differential Personality Inventory.

4. Self-Efficacy Scale (SES): A scale designed to measure one's beliefs about their own competency. The scale consists of 30 items and subjects are to respond to each item statement by indicating whether they disagree strongly or moderately, agree moderately or strongly, or neither agree nor disagree with each statement according to how well they feel each statement describes them. Examples of statements include: "I avoid trying to learn new things when they look too difficult for me," and "There is some good in everybody". The Self Efficacy scale (SES) yields a total self efficacy score for each subject. The internal consistency coefficient for the SES is .86.

5. Wallston's Multidimensional Health Locus of Control (MHLC): These scales were designed to measure the locus of control of health-related behavior. It has previously been used to assess psychosocial factors in diabetics (Peyrot & McMurry, 1985). The scale consists of 15 item statements, and subjects are asked to respond to each item statement by indicating on a five point scale how well they feel each statement describes them. Examples of statements include: "People in my family have confidence in me", "People who know me trust me and respect me", and "I share the same approach to life that my friends do". Subjects respond with a "1" if they feel the statement describes them very much, with a "2" if they feel it describes them quite well, a "3" if they feel it describes them somewhat, a "4" if it does not describe them very well and a "5" if they feel the statement does not describe them at all. The MHCL yields three locus of control scores for each individual subject: an internal health locus of control, a chance health locus of control, and a powerful others health locus of control. Therefore, this scale is believed to indicate where the individual places responsibility for his or her own health, and for the control of health. Internal consistency coefficients for the MHCL range from .83 to .86 and the individual scales correlate well with other measures of

locus of control.

Ethical Considerations

All ethical rights of the patients were conserved to the fullest extent. Patients were asked to sign a written consent form indicating that they understood conditions of the research and what was required of them. The consent form also allowed the investigators to gain access to the information in the patients' medical records. Subjects were informed that they were free to withdraw from the study at any time if they so desired. Confidentiality was promised by all those persons involved in the research. All of the subjects' responses and any personal information gained from the participants as a result of this research was kept in the strictest confidence. If requested, patients were sent a letter informing them of the results of their own personal protocols. Any person scoring greater than two standard deviations above normal on either the anxiety or depression scales of the Jackson Basic Personality Inventory were sent a form letter explaining to them that it is realized that people often have difficulty coping with diabetes. Free counselling services were offered to these subjects through the University of Alberta Education Clinic. In addition, the diabetologist at the Edmonton General Hospital was given the names of the individuals who were sent these letters so that he would be

aware of the difficulties that these patients were experiencing. At the completion of the study, letters were mailed to each participant thanking them for their participation in the study and explaining some of the results of the research.

Analysis of Data

All protocols were scored according to the directions provided in their respective manuals. Secondary complications, control and obesity were coded by the nurses at the clinic on a 3 point scale according to degree of severity (see appendix). Treatment for each subject was coded as either insulin-treated or non-insulin treated.

Pearson correlations were performed for each diabetic type and duration group comparing each of the individual variables with mean scores. Means and standard deviations for each variable and for each diabetic category were tabulated. In addition, Pearson correlations were performed for each variable comparing the total diabetic group scores. In order to compare the Type I, Onset I and Type I, Onset II groups, T tests were performed to determine whether any significant differences existed between the group means with respect to the many variables. However, the small size of the Type I, Onset I group makes the interpretation of such statistical data likely to be lacking in accuracy. Nevertheless, the

results of statistical analysis and a description of the data for these groups are provided. The Type II, Onet I and Type II, Onset II groups were compared via Multivariate Analysis of Variance testing. As there are a very large number of variables involved in a study of this nature, interpretation and discussion of the data results is limited to those findings which appear to be most relevant based on the preceding literature review. In particular, special attention is given to the following variables: scores obtained on the Jackson Basic Personality Inventory with emphasis on the anxiety and depression scales; glycosylated hemoglobin levels; secondary complications including neuropathy, nephropathy, vascularity, and retinopathy; Campbell's Indexes of Wellbeing, and the Physical, and Psychosocial Scales of the Sickness Impact Profile.

IV. RESULTS

Introduction

First, the sample characteristics of the four diabetic groups will be described. Second, tables describing the means, standard deviations, group data, and descriptions of variables are provided. Third, statistical comparisons between Type II, Onset I and Type II, Onset II are made with respect to the psychological characteristics provided by the Jackson Basic Personality Inventory and with respect to the physiological variables. Results of these comparisons are then discussed. Fourth, statistical comparisons between Type I, Onset I, and Type I, Onset II are made with respect to the psychological characteristics provided by the Jackson Basic Personality Inventory, and the physiological variables. Results of these comparisons are then discussed. Fifth, frequency tables are provided for Type I and Type II diabetics with respect to the presence and severity of secondary complications. This is followed by a comparative discussion of the incidence and severity of secondary complications found in Type I and Type II diabetics. Sixth, frequency tables including the rates of abnormally high levels of anxiety and depression in Type I and Type II is presented and discussed. Seventh, each of the research questions which were presented in Chapter 1 are answered separately on the basis of this study's findings. Finally, correlational tables are

provided with respect to numerous variables. (A complete listing of all correlations conducted between the variables are provided in the Appendix).

SAMPLE CHARACTERISTICS

There were 136 diabetic subjects who participated in this study. Of the total number of subjects, 79 were males and 57 were female diabetics. All subjects who participated in this study were attending a diabetic outpatient program at the Edmonton General Hospital. 55% of the subjects were present for a six month follow-up examination; 45% were present for a four day educational program. Seven of the subjects were recently diagnosed Type I diabetics (Type I, Onset I); thirty-seven of the subjects were long duration Type I diabetics (Type I, Onset II); thirty-four of the subjects were recently diagnosed Type II diabetics (Type II, Onset I), and fifty-eight of the subjects were long duration Type II diabetics (Type II, Onset II). The ages of all subjects ranged from 16 to 86 years, with a mean of 51, and standard deviation of 17. More than 25% of the subjects lived outside the Edmonton area, and 23% of the subjects were retired. The HbAlc for the total group ranged from a low of 3.4 to a high of 15.2 with a mean of 7.5, and a standard deviation of 2.1. Secondary complications were evident in more than half of the diabetic subjects (51%), and secondary complications in the 'severe' category were observed in 20% of the subjects.

TABLE 1

MEANS AND STANDARD DEVIATIONS FOR THE

THE TOTAL DIABETIC SAMPLE

N= 136

VARIABLE:	MEAN:	STANDARD DEVIATION:
AGE	51.13	17.21
DURATION	11.47	9.43
HbA1c	7.47	2.10
CAMPBELL'S SCALE OF WELLBEING:		
Ca9	5.10	1.45
AFFECT	5.40	1.26
WELLBEING	11.04	2.70
WALLSTON'S MULTIDIMENSIONAL HEALTH LOCUS OF CONTROL:		
IHLC	27.38	4.70
POLC	21.19	6.01
CHLC	15.32	6.49
THE SELF EFFICACY SCALE:		
SES	83.12	14.06

JACKSON BASIC PERSONALITY INVENTORY:

SCALE:	MEAN:	STANDARD DEVIATION:
Hypochondriasis	7.07	4.43
Depression	4.30	4.55
Denial	7.55	3.42
Interpersonal Problems	7.86	3.92
Alienation	3.70	2.90
Persecutory Ideas	5.04	3.09
Anxiety	7.00	4.25
Thinking Disorder	3.31	2.87
Impulse Expression	5.80	3.98
Social Introversion	5.10	3.40
Self-Depreciation	2.54	2.94
Deviation	3.09	3.14

SICKNESS IMPACT PROFILE:

SCALE:	MEAN:	STANDARD DEVIATION:
Sleep	.15	.18
Emotional Behavior	.10	.16
Bodycare and Movement	.03	.06
Home Management	.26	.10
Mobility	.04	.09
Social Interaction	.11	.16
Ambulation	.06	.11
Alertness	.15	.23
Communication	.05	.10
Work	.08	.20
Recreation	.15	.21
Eating	.06	.06
Physical SIP	15.24	24.90
Psychosocial SIP	37.54	51.07
Total SIP	91.16	89.12

TABLE 2

MEANS AND STANDARD DEVIATIONS
FOR EACH DIABETIC TYPE, ONSET GROUP

VARIABLE:	Type I Onset I	Type I Onset II	Type II Onset I	Type II Onset II
Mean (Std.Dev.)				
Age	38.71 (19.39)	34.76 (13.72)	53.47 (13.70)	61.71 (11.16)
Duration (in yrs.)	.00 (0.00)	17.27 (7.80)	.00 (0.00)	15.88 (6.00)
HbA1c	7.92 (3.36)	8.19 (2.12)	6.28 (1.39)	7.63 (2.04)
CAMPBELL'S SCALE OF WELLBEING:				
Ca 9	5.83 (.75)	4.89 (1.56)	5.12 (1.43)	5.14 (1.43)
AFFECT	6.27 (0.60)	5.14 (1.49)	5.46 (1.17)	5.42 (1.18)
WELLBEING	12.78 (1.17)	10.52 (3.41)	11.14 (2.47)	11.12 (2.55)
WALLSTON'S MULTIDIMENSIONAL LOCUS OF CONTROL:				
IHLC	26.14 (6.82)	27.81 (4.39)	27.35 (4.87)	27.27 (4.61)
POLC	25.29 (6.65)	19.60 (5.88)	21.06 (5.83)	21.81 (5.95)
CHLC	19.43 (6.32)	13.73 (5.59)	15.59 (7.16)	15.70 (6.50)
THE SELF EFFICACY SCALE:				
SES	92.86 (8.40)	82.60 (14.99)	82.59 (14.21)	82.57 (13.76)

JACKSON BASIC PERSONALITY INVENTORY:

Mean (Std.Dev.)	Type I Onset I	Type I Onset II	Type II Onset I	Type II Onset II
SCALE:				
Hypochondriasis	3.86 (3.89)	6.24 (5.19)	6.30 (4.03)	8.50 (3.76)
Depression	1.71 (.95)	5.51 (5.35)	4.49 (4.32)	3.71 (3.89)
Denial	6.86 (2.91)	6.30 (2.50)	8.15 (3.55)	8.13 (3.74)
Interpersonal Problems	9.14 (4.53)	8.24 (4.40)	8.06 (3.41)	7.31 (3.81)
Alienation	3.29 (1.60)	3.78 (3.69)	3.88 (2.64)	3.58 (2.61)
Persecutory Ideas	3.71 (1.11)	5.08 (3.26)	5.21 (3.19)	5.07 (3.10)
Anxiety	5.14 (2.80)	7.68 (4.88)	6.55 (4.08)	6.95 (4.03)
Thinking Disorder	2.14 (.90)	2.84 (3.18)	3.91 (2.98)	3.42 (2.70)
Impulse Expression	6.57 (4.32)	6.24 (3.59)	6.00 (4.16)	5.27 (4.12)
Social Introversion	4.57 (4.08)	5.70 (4.49)	5.24 (2.49)	4.66 (2.94)
Self-Depreciation	1.000 (1.41)	3.22 (4.33)	2.27 (2.24)	2.44 (2.16)
Deviation	1.71 (1.25)	3.76 (3.91)	2.81 (2.97)	2.98 (2.79)

SICKNESS IMPACT PROFILE:

Mean (Std.Dev.)	Type I Onset I	Type I Onset II	Type II Onset I	Type II Onset II
SCALE:				
Sleep	.02 (.04)	.11 (.13)	.14 (.18)	.20 (.20)
Emotional Behavior	.07 (.12)	.13 (.21)	.08 (.16)	.10 (.13)
Bodycare & Movement	.01 (.03)	.02 (.04)	.01 (.02)	.06 (.08)
Home Management	.27 (.04)	.24 (.10)	.22 (.06)	.29 (.12)
Mobility	.01 (.03)	.05 (.08)	.01 (.04)	.07 (.11)
Social Interaction	.10 (.15)	.16 (.22)	.09 (.18)	.09 (.11)
Ambulation	.01 (.04)	.03 (.09)	.02 (.05)	.11 (.13)
Alertness	.14 (.34)	.17 (.26)	.13 (.25)	.16 (.18)
Communication	.04 (.08)	.07 (.13)	.02 (.05)	.05 (.10)
Work	.13 (.31)	.12 (.24)	.03 (.09)	.09 (.20)
Recreation	.19 (.19)	.15 (.24)	.06 (.12)	.19 (.22)
Eating	.06 (.05)	.06 (.06)	.07 (.06)	.05 (.06)

SICKNESS IMPACT PROFILE:

Mean (Std.Dev.)	Type I Onset I	Type I Onset II	Type II Onset I	Type II Onset II
SCALE:				
Physical SIP	4.16 (7.96)	9.43 (18.50)	4.61 (8.24)	26.50 (31.20)
Psychosocial SIP	33.33 (44.27)	49.70 (67.23)	26.71 (54.34)	36.65 (35.79)
Total SIP	72.21 (72.90)	93.57 (109.22)	61.91 (73.32)	109.03 (82.54)

TABLE 3. SUMMARY OF GROUP CHARACTERISTICS

Type I, Onset I:	N= 7
4 Females; 3 Males:	
Age Range from:	16 to 68 years
HbAlc Range from:	4.8 to 13.8
Secondary Complications:	none noted in this group
Type I, Onset II:	N= 37
21 Females; 16 Males:	
Age Range from:	17 to 71 years
HbAlc Range from:	3.4 to 15.2
Secondary Complications:	noted in 70% of the subjects; of the 'severe' category noted in 35% of the subjects.
Type II, Onset I:	N= 34
23 Females; 11 Males:	
Age Range from:	27 to 78 years
HbAlc Range from:	3.6 to 10.3
Secondary Complications:	noted in 50% of the subjects; of the 'severe' category noted in 9% of the subjects.
Type II, Onset II:	N= 58
32 Females: 26 Males:	
Age Range from:	38 to 86 years
HbAlc Range from:	4.7 to 11.8
Secondary Complications:	noted in 71% of the subjects; of the 'severe' category noted in 31% of the subjects.
Total Male Subjects:	N= 79
Total Female Subjects:	N= 57
Total Diabetic Subjects:	N= 136
Total Age Range from:	16 to 86 years
Total HbAlc Range from:	3.4 to 15.2
Secondary Complications:	noted in 51% of the subjects; of the 'severe' category noted in 20% of the subjects.

TABLE 4.

ABBREVIATIONS AND DESCRIPTION OF VARIABLES

VARIABLE:	DESCRIPTION:
Type	Type of Diabetes (I or II)
Onset	Onset I (recently diagnosed - within the last 1 year) Onset II (long term - diagnosis 10 or more years previous).
Age	Age of Subject
Sex	Sex of Subject
Duration	Length of time diabetic (in years)

PHYSIOLOGICAL MEASURES

Scored as 0, 1 or 2 based on degrees of severity. From low to increasing severity. Highest severity = 2. Lowest severity = 0. See appendix for details.

VARIABLE:	DESCRIPTION:
Control	Degree of diabetic control
Obesity	Degree of Obesity
Vascular	Vascularity. Severity of Vascular pathology.
Nupathy	Neuropathy. Severity of Neuropathology.
Nppathy	Nephropathy. Severity of Nephropathology.
Rtpathy	Retinopathy. Severity of Retinopathy.
HbAlc	Blood Glucose Levels as indicated by Glycosylated Hemoglobin Assay.

CAMPBELL'S SCALES OF WELLBEING

VARIABLE:	DESCRIPTION:
Ca 9	Score on the Campbell's 9th scale which indicates Overall Life Satisfaction. High scores indicative of greater satisfaction with life. Lowest possible score = 1 Highest possible score = 7
Affect	Score on Campbell's Index of General Affect. Score is comprised of the total mean for scores on Campbell's scales 1 through 8. High scores indicative of positive affect. Lowest possible score = 1 Highest possible score = 7
Wellbeing	Score is comprised of the scores on Campbell's Index of General Affect and Ca9: (1.1 x Affect) + Ca9. Higher scores indicative of greater wellbeing. Lowest possible score = 2.1 Highest possible score = 14.70

THE SELF EFFICACY SCALE

SES	Higher scores indicative of greater self efficacy. Lowest possible score = 23 Highest possible score = 115

WALLSTON'S HEALTH LOCUS OF CONTROL

IHLC	Internal Health Locus of Control. Higher scores indicative of higher degree of an internal health locus of control. Lowest possible score = 6 Highest possible score = 36

WALLSTON'S HEALTH LOCUS OF CONTROL

VARIABLE:	DESCRIPTION:
POLC	Powerful Others Health Locus of Control. Higher scores indicative of higher degree of powerful others health locus of control. Lowest possible score = 6 Highest possible score = 36
CHLC	Chance Health Locus of Control. Higher scores indicative of higher degree of chance health locus of control. Lowest possible score = 6 Highest possible score = 36

JACKSON'S BASIC PERSONALITY INVENTORY

HYP	HYPOCHONDRIASIS
DEP	DEPRESSION
DEN	DENIAL
INP	INTERPERSONAL PROBLEMS
ALN	ALIENATION
PID	PERSECUTORY IDEAS
ANX	ANXIETY
THD	THINKING DISORDER
IME	IMPULSE EXPRESSION
SOI	SOCIAL INTROVERSION
SDP	SELF DEPRECIATION
DEV	DEVIATION

Highest possible score on each scale = 20. Higher scores indicative of a greater degree of the corresponding personality characteristic. Lower scores indicative of a greater degree of the opposite corresponding personality characteristic.

SICKNESS IMPACT PROFILE

SLEEP	SLEEP AND REST	7 items
EMOTION	EMOTIONAL BEHAVIOR	9 items
BODYCARE	BODYCARE AND MOVEMENT	23 items
HOME	HOME MANAGEMENT	10 items
MOBILE	MOBILITY	10 items
SOCIAL	SOCIAL INTERACTION	20 items
AMBUL	AMBULATION	12 items
ALERT	ALERTNESS	10 items
COMMUN	COMMUNICATION	9 items
WORK	WORK	9 items
RECREAT	RECREATION	8 items
EATING	EATING	9 items

PSYCHSIP	PSYCHOSOCIAL SICKNESS IMPACT PROFILE (Calculated from scores obtained on Social Interaction, Communication, Alertness Behavior, and Emotional Behavior Scales).
PHYSSIP	PHYSICAL SICKNESS IMPACT PROFILE (Calculated from scores obtained on Ambulation, Mobility, and Bodycare and Movement Scales).
TOTSIP	TOTAL SICKNESS IMPACT PROFILE (Calculated from scores obtained on all 12 scales). Encompasses both the physical and psychosocial sickness impact profiles.

SAMPLE TEST SCORES - SUMMARY DATA

According to findings on the Jackson Basic Personality Inventory (see Table 1), diabetics tended to score highest on the Hypochondriasis (mean 7.07), Denial (mean 7.55), Interpersonal Problems (mean 7.86), and Anxiety (mean 7.00) scales. Lower mean scores were observed on the Self-depreciation (mean 2.54), Deviation (mean 3.09), Thinking Disorder (mean 3.31), and Alienation (mean 3.70) scales of the Jackson Basic Personality Inventory. Higher scores on the Hypochondriasis scale suggests that diabetics may be more troubled by physical symptoms and may have a tendency to express more complaints of a somatic nature. Considering that secondary complications were present in more than half of the participants, it is not unexpected to find these subjects expressing more concern and complaints in relation to their health status. The tendency to score higher on the Anxiety, and Interpersonal problems scales, suggests that diabetics may become easily frustrated, annoyed, and overly excited about minor events or changes. Considering the importance of maintaining glycemic control, it is not surprising to find diabetics worrying or becoming easily upset about slight changes in their glycosylated hemoglobin levels. A high score on the Denial scale suggests that one of the ways that diabetics may attempt to cope with their illness is to avoid

discussing or dwelling on it. Finally, the tendency for diabetics to score lower on the Self-depreciation, Deviation, Thinking Disorder and Alienation scales, indicates that the participants of this study generally felt pretty good about themselves, and considered themselves capable of functioning in life very much the same as other people in society.

Results from Wallston's Multidimensional Health Locus of Control, indicated that an Internal Health Locus of Control was favored most by the respondents (mean 27.38), and a Chance Health Locus of Control was favored least (mean 15.32). According to these findings, the average diabetic is more likely to feel they are responsible for the control of their own health, and are less likely to leave their diabetic control up to chance.

Scores obtained on the Sickness Impact Profile indicated that the areas most likely to be reported as negatively effected by diabetes include: Sleep (mean .15), Home Management (mean .26), and Recreation and Pastimes (mean .15). In addition, psychosocial functioning was perceived by the participants as being interfered with more as a consequence of their diabetes than physical functioning (mean 37.54 and 15.24 respectively).

Type I, Onset I Group

Comprised of only seven subjects, the recently

diagnosed Type I diabetics presented as the smallest group of diabetics observed at the clinic. This is not too surprising, given the following. First, Type I diabetes is a far less common type of diabetes in comparison to Type II diabetes (approximately 30% of all diabetics are Type I). Second, most Type I diabetics are diagnosed very early in life. As this study dealt only the adult population (ages 16 and over), it is not unexpected to find relatively few recently diagnosed adults.

The Type I, Onset I group was comprised of four males and 3 females. Their ages ranged from 16 to 68 years of age, with a mean age of 38 years, and a standard deviation of 19 years. The majority of the subjects were under the age of 40 (five subjects), with two over 60 years of age. Five of the subjects were employed; one was a student; and one was retired. Glycemic control for this group ranged from a low of 4.8 to a high of 13.8 (mean = 7.9; standard deviation 3.4). No secondary complications were observed in any of the subjects belonging to this group.

Caution must be used in interpreting the data and results provided by the Type I, Onset I group owing to its very small size. Unfortunately, the comparatively small size of this group makes appropriate statistical analysis very difficult to conduct and interpret, and the likelihood

of type one errors arising is increased considerably. Furthermore, the small size of this group leaves a strong possibility open that this group is not very representative of all recently diagnosed Type I diabetics. Consequently, extreme caution is warranted when arriving at any solid conclusions based on the findings of the Type I, Onset I group used in this study. Any interpretations made are tentative and subject to correction.

According to the results presented in Table 2, the recently diagnosed Type I group scored on the average, higher on all scales of Campbell's Index of Wellbeing in comparison to the other three groups. The Type I, Onset I group is shown as having the highest group mean for overall life satisfaction (5.83), positive affect (6.27), and general wellbeing (12.78) in comparison to the other three groups. In addition, the Type I, Onset I group shows the highest mean score on the Self-Efficacy scale (92.86) in comparison to the other three groups. These findings suggest that the recently diagnosed Type I group generally seems quite happy and satisfied with their lives. Their diabetes seems to have little negative impact as to how they feel about themselves or about life in comparison to the other three groups. Furthermore, the Type I Onset I group was shown to have the lowest overall mean score on several scales of the Jackson Basic Personality Inventory

in comparison to the other three groups. Recently diagnosed Type I diabetics on the average scored lower on the Hypochondriasis, Depression, Alienation, Persecutory Ideas, Thinking Disorder, Anxiety, Self-depreciation and Deviation scales when compared with the other three groups. These findings suggest that psychologically, the recently diagnosed Type I group seemed to fair better than the other three groups. They appeared to experience little difficulty with negative emotional or thinking states, and expressed fewer complaints of a somatic nature. However, in comparison to the other three groups, the recently diagnosed Type I subjects tended to score higher on the Interpersonal Problems scale of the Jackson Basic Personality Inventory. This suggests that this group, in comparison to the other three groups, is more likely to get easily annoyed and upset by little irritations and inconveniences.

According to the results on the Sickness Impact Profile, the recently diagnosed Type I group scored higher on the average on the Work scale (mean .13) in comparison to the other three groups. Therefore, this group perceived their diabetes as having a negative impact on their Work life. Recently diagnosed Type I diabetics also scored lower, (on the average) on the Physical scale of the Sickness Impact Profile (mean 4.16) in comparison to the

other three groups. This suggests that this group felt their diabetes had less negative impact upon their physical functioning than the other three groups.

Type I, Onset II Group

This group consisted of 16 males and 21 females with ages ranging from 17 to 71 years of age (mean 35; standard deviation 14). Almost half of this group were presently employed. Four were students; seven were housewives; three were retired; and five were not working for various reasons. This group included both the overall low and the overall high HbAlc reading for the total four groups. Glycemic control ranged from a low of 3.4 to a high of 15.2 (mean HbAlc 8.2; standard deviation of 2.1). Secondary complications were evident in over 70% of the subjects in this group and 35% had secondary complications considered to be in the 'severe' category. Severe neuropathy was observed in seven of the subjects; severe nephropathy was observed in two of the subjects; and severe retinopathy was observed in six of the subjects.

According to the results on Table 2, the long duration Type I group scored the highest overall mean in terms of diabetic duration (17.27) and HbAlc level (8.19). In addition, this group was shown to have a high incidence of secondary complications (70%). Therefore, it appears that on the average, the long duration Type I diabetics seemed

to have the highest rate of diabetic morbidity in comparison to the other three grcups. In addition, these results point to a potential association between diabetic duration, elevated glycosylated hemoglobin levels, and the development of secondary complications.

According to Table 2, the Type I, Onset II group scored lower on all three of Campbell's scales of Wellbeing in comparison to the other three groups. As such, this group is in sharp contrast to the Type I, Onset I group. In addition, the long duration Type I group scored higher on several scales of the Jackson Basic Personality Inventory. Higher average scores were noted on the Depression (mean 5.51), Anxiety (mean 7.68), Social Introversion (mean 5.70), Self-depreciation (mean 3.22), and Deviation (mean 3.76) scales, in comparison to the other three groups. These findings suggest that Type I, Onset II diabetics are more likely to find that their diabetes has a negative impact upon their personality and psychological functioning. In addition, the long duration Type I group scored higher on the Emotional Behavior (mean .13), Social Interaction (mean .16), Alertness (mean .17), and Psychosocial (mean 49.70) scales of the Sickness Impact Profile, in comparison to the other three groups. Furthermore, long duration Type I diabetics seemed to perceive their illness having greater negative impact on

their psychosocial functioning in comparison to their physical functioning.

Type II, Onset I Group

11 males and 23 females between the ages of 27 and 78 comprised the recently diagnosed Type II group (mean age 54; standard deviation 14). Over half of the subjects controlled their diabetes through the use of diet alone (56%). Eight of the subjects took insulin, and seven of the subjects took pills in order to help control their diabetes. The majority of the subjects were presently employed and working (59%); seven were housewives; six were retired; and one subject was presently not working. The HbAlc for this group ranged from a low of 4.7 to a high of 11.8 (mean 6.3; standard deviation 1.4). Therefore, this group had the lowest HbAlc mean in comparison to the other three groups. The presence of secondary complications were evident in 50% of the subjects. Three subjects were observed to have the 'severe' secondary complication of neuropathy. All other secondary complications observed were of a lesser degree of severity.

Type II, Onset II Group

The long duration Type II group comprised the largest and eldest group of diabetic subjects seen at the clinic in comparison to the other three groups. The Type II, Onset II group consisted of 26 males and 32 females

ranging in ages from 38 to 86 years (mean age 62; standard deviation 11). Fourty-seven of the subjects (81%) were presently taking insulin to assist them in controlling their diabetes. 35% of the subjects were presently employed and working. 29 were retired; 9 were housewives, and 2 were students. Glycemic control for this group ranged from a low of 4.7 to a high of 11.8 (mean 7.6; standard deviation 2.0). Secondary complications were evident in 71% of the subjects and 31% were observed to have secondary complications of the 'severe' category. Neuropathy was evident in all cases of severe secondary complications. Two subjects with severe neuropathy also had severe nephropathy; two subjects with severe neuropathy also had severe retinopathy; and one subject was observed to have a combination of severe neuropathy, nephropathy, and retinopathy.

COMPARISON OF PSYCHOLOGICAL CHARACTERISTICS IN TYPE II, ONSET I and TYPE II, ONSET II DIABETICS

A 2 x 12 multivariate analysis of variance did not reveal any overall significant difference between recently diagnosed and long duration Type II diabetics with respect to the scores that were obtained on the twelve scales of the Jackson Basic Personality Inventory, [$F(1,85) = 1.50$, $p > .05$ (see Table 5)]. However, a univariate analysis of variance revealed a significant difference with respect to

the Hypochondriasis scale [$F(1,85) = 5.74$, $p < .05$]. An examination of the means, indicates that long duration Type II diabetics (scoring higher on the Hypochondriasis scale), were found to have more complaints and concerns regarding their health and physical functioning. This is not surprising given that the Onset II group was, by comparison to the Onset I group, an older population experiencing a higher incidence of secondary complications. Therefore, the somatic complaints and concerns expressed by the long duration Type II diabetics are likely legitimate considering their physical status.

TABLE 5.

Comparison of Type II, Onset I and Type II, Onset II and Scores on the scales of the Jackson Basic Personality Inventory

VARIABLE	TYPE II ONSET I n= 34	TYPE II ONSET II n= 58	F	P
Hypochondriasis	6.30	8.49	5.74	.02*
Depression	4.49	3.71	.54	.46
Denial	8.15	8.13	.00	.97
Interpersonal Problems	8.01	7.31	1.17	.28
Alienation	3.88	3.58	.04	.85
Persecutory Ideas	5.21	5.07	.00	.99
Anxiety	6.55	7.00	.13	.72
Thinking Disorder	3.91	3.42	.60	.44
Impulse Expression	6.00	5.27	.93	.34
Social Introversion	5.24	4.66	.92	.34
Self-Depreciation	2.27	2.44	.15	.70
Deviation	2.81	3.00	.07	.79

* $p < .05$

TABLE 6.

Comparison of Type II, Onset I and Type II, Onset II and Physiological Variables

VARIABLE	TYPE II ONSET I n= 34	TYPE II ONSET II n= 58	F	P
Vascularity	.26	.46	2.20	.14
Neuropathy	.35	.98	9.68	.00*
Nephropathy	.04	.39	6.94	.01*
Retinopathy	.00	.22	3.51	.07
HbAlc	6.42	7.48	4.94	.03*

* $p < .05$

COMPARISON OF PHYSIOLOGICAL VARIABLES IN TYPE II, ONSET I AND TYPE II, ONSET II DIABETICS

A 2 x 5 multivariate analysis of variance revealed overall significant differences between the Type II, Onset I and Type II, Onset II groups with respect to the physiological variables [$F(1,67) = 3.14$, $p < .05$ (see Table 6)]. Furthermore, univariate analysis of variance revealed significant differences between the groups with respect to Neuropathy $F(1,67) = 9.68$, Nephropathy $F(1,67) = 6.94$, and HbAlc $F(1,67) = 4.94$, $p < .05$. This indicates that long duration Type II diabetics are more likely to have secondary complications of Neuropathy, and Nephropathy in comparison to recently diagnosed Type II diabetics. In addition, long duration Type II diabetics are more likely to have higher glycosylated hemoglobin levels than recently diagnosed Type II diabetics. These findings are consistent with research which has revealed an association between diabetic duration and the presence of secondary complications, and between poor metabolic control and the development of secondary complications.

COMPARISON OF PSYCHOLOGICAL CHARACTERISTICS IN TYPE I, ONSET I AND TYPE I, ONSET II DIABETICS

A comparison of the Type I means with respect to the scores on the Jackson Basic Personality Inventory was conducted using Hoetelling's t-test. Results

revealed significant differences between recently diagnosed and long duration Type I diabetics on four scales (see Table 7). Long duration Type I diabetics were found to score significantly higher on the Depression, Persecutory Ideas, Self-depreciation, and Deviation scales, in comparison to recently diagnosed Type I diabetics. These findings indicate that across time, Type I-diabetics are likely to develop difficulties in certain areas of psychological functioning. Such difficulties include: tendencies towards feelings of self-inadequacy or self-worthlessness; perceptions that others are trying to harm them or make things more difficult for them; and unusual behavior or pathological thinking patterns. However, caution must be used in interpreting the results of the t-test conducted with these groups. The small size of the Type I, Onset I group increases the chances of a making a type one error considerably.

COMPARISON OF PHYSIOLOGICAL VARIABLES IN TYPE I, ONSET I AND TYPE I, ONSET II DIABETICS

A comparison of the Type I means with respect to the the physiological variables was conducted used Hoetelling's t-test. Results revealed significant differences between the two groups with respect to the incidence and severity of the secondary complication of Retinopathy (see Table 8).

TABLE 7

Comparison of Mean Scores on the Jackson Basic Personality Inventory for Type I Diabetics

VARIABLE	TYPE I ONSET I n =7	TYPE I ONSET II n =37	T	P
Hypochondriasis	3.86	6.24	-1.15	0.26
Depression	1.71	5.51	-4.00	0.00*
Denial	6.86	6.30	0.53	0.60
Interpersonal Problems	9.14	8.24	0.49	0.62
Alienation	3.29	3.78	-0.58	0.57
Persecutory Ideas	3.71	5.08	-2.01	0.05*
Anxiety	5.14	7.68	-1.32	0.19
Thinking Disorder	2.14	2.84	-1.11	0.27
Impulse Expression	6.57	6.24	0.21	0.83
Social Introversion	4.57	5.70	-0.62	0.54
Self-Depreciation	1.00	3.22	-2.49	0.02*
Deviation	1.71	3.76	-2.56	0.02*

*p <.05

TABLE 8

Comparison of Mean Scores on the Physiological Variables for Type I Diabetics

VARIABLE	TYPE I ONSET I n =7	TYPE I ONSET II n =37	T	P
Vascularity	0.00	0.06	-0.59	0.16
Neuropathy	0.00	0.63	-1.89	0.07
Nephropathy	0.00	0.23	-1.01	0.32
Retinopathy	0.00	0.75	-2.48	0.02*
HbAlc	7.92	8.19	-0.25	0.81

*p <.05

Although average increases across time were noted for Vascularity, Neuropathy, Nephropathy, and glycosylated hemoglobin levels, only the rate and severity of Retinopathy was found to increase significantly. These findings are consistent with research which has indicated an association between Type I diabetes, diabetic duration and the development of secondary complications. More specifically, retinopathy seems to be a common secondary complication found in long duration Type I diabetics and research has suggested that the development of retinopathy may be associated with episodes of hyperglycemia. Again however, caution is advised in interpreting the results of statistical tests conducted with the recently diagnosed Type I group due to its small size, and the strong possibility of type one errors.

SECONDARY COMPLICATIONS - INCIDENCE AND SEVERITY

Frequency analysis reveals a higher frequency of secondary complications in the long duration diabetics in comparison to the recently diagnosed diabetics (see Table 9). Retinopathy is revealed to be the most frequent secondary complication found in the long duration Type I group, while neuropathy is found to be the most frequent 'severe' secondary complication found in the long duration Type II group. As no incidence of secondary complication was noted in the recently diagnosed Type I group, the

TABLE 9

FREQUENCIES OF SECONDARY COMPLICATIONS

SECONDARY COMPLICATIONS:

0 to 2: lesser to more severe pathology

VARIABLE:	Type I Onset I n= 7 Frequency	Type I Onset II n= 37 Frequency	Type II Onset I n= 34 Frequency	Type II Onset II n= 58 Frequency
VASCULARITY				
Value				
0	6	33	22	31
1	0	2	8	20
2	0	0	0	4
N/A	1	2	4	6
NEUROPATHY				
Value				
0	6	20	20	19
1	0	8	7	16
2	0	7	3	17
N/A	1	2	4	6
NEPHROPATHY				
Value				
0	6	29	25	35
1	0	4	3	12
2	0	2	0	3
N/A	1	2	6	8
RETINOPATHY				
Value				
0	6	15	27	44
1	0	15	0	5
2	0	6	0	3
N/A	1	1	7	6

N/A - data not available

results from this data suggest significant detrimental physiological changes occur across the diabetic lifespan, particularly for the Type I diabetic. With the Type II group, neuropathy is shown as being the most commonly found secondary complication. Furthermore, neuropathy is also found to be the most frequently occurring 'severe' secondary complication in the Type II group. No incidence of 'severe' secondary complications of vascularity, nephropathy, or retinopathy are found in the recently diagnosed Type II group. However, a higher rate of secondary and 'severe' secondary complications is noted in the long duration Type II diabetic in comparison to the recently diagnosed Type II diabetic. The overall findings of frequency analysis point to an association between diabetic duration and increased incidence and severity of secondary complications. In addition, there appears to be notable differences between the two diabetic Type groups in terms of proneness to types of secondary complications. 50% of the long duration Type I diabetics showed signs of retinopathy, 37% showed signs of neuropathy, 15% showed signs of nephropathy, and 5% showed signs of vascularity. In comparison, 18% of the long duration Type II diabetics showed signs of retinopathy, 52% showed signs of neuropathy, 23% showed signs of nephropathy, and 39% showed signs of vascularity. Therefore, Type I diabetics seem

more likely than Type II diabetics to develop retinopathy, and Type II diabetics seems more likely than Type I diabetics to develop neuropathy, nephropathy, or vascular pathology over time.

THE INCIDENCE OF ABNORMALLY HIGH LEVELS OF DEPRESSION AND ANXIETY IN TYPE I AND TYPE II DIABETICS.

Frequency analysis of scores obtained on the Anxiety and Depression scales of the Jackson Basic Personality Inventory reveals a higher incidence of severe depression and anxiety in the long duration Type I diabetics when compared to the other three groups (see Table 10). Furthermore, an increased incidence of severe anxiety (but not depression), is noted across time for the Type II diabetics. In view of the differences between these two diabetic types with respect the incidence of secondary complications, an association between the presence of secondary complications and anxiety/depression is suspect. Long duration Type I and Type II diabetics are very similar in terms of the incidence of secondary complications (70% and 71% respectively), and in terms of the incidence of 'severe' secondary complications (35% and 30% respectively). However, where these two groups differ is with respect to the types of secondary complications that they are more likely to develop. Type I diabetics seem more predisposed to developing retinopathy, and Type II

diabetics seem more likely to develop vascular problems, neuropathy or nephropathy. An examination of the individual protocols showed that of the Type I long duration diabetics who scored high on depression/anxiety, all but two showed signs of retinopathy. These findings are consistent with research indicating a link between depression and retinopathy. Furthermore, over half of the long duration Type II diabetics who showed signs of more than one type of secondary complication, also scored very high on the depression/anxiety scales. Therefore, high rates of depression and anxiety appear to be associated with the presence of secondary complications in Type I and II diabetics.

TABLE 10

NUMBER OF INDIVIDUALS SCORING HIGH ON THE DEPRESSION AND ANXIETY SCALES OF THE JACKSON BASIC PERSONALITY INVENTORY

High = Scoring two or more standard deviations above the mean.

	TYPE I ONSET I	TYPE I ONSET II	TYPE II ONSET I	TYPE II ONSET II
DEPRESSION	0	8 (22%)	3 (9%)	4 (7%)
ANXIETY	0	6 (16%)	2 (6%)	5 (9%)

RESEARCH QUESTIONS ANSWERED

1. How does diabetes mellitus effect the social, psychological and physical aspects of life for diabetics both initially, and after they have lived with it for several years?

According to results from this study, the social, psychological, and physical effects of diabetes mellitus are strongly influenced by degree of metabolic control, by the presence and severity of secondary complications, and by the degree of physiological changes that occur over time. Glycosylated hemoglobin levels are found to increase over time in both Type I and Type II diabetics (see Tables 6 and 8). In the Type I group, good metabolic control is found to be associated with lower rates of nephropathy, and higher levels of life satisfaction and feelings of general wellbeing (see Table 13). In stark contrast to these findings, results show that higher glycosylated hemoglobin levels in Type II diabetics are to related to lower scores on the Hypochondriasis and Depression scales of the Jackson Basic Personality Inventory (see Table 12). Therefore, Type I diabetics seem to feel better when their glycosylated hemoglobin levels are lower, while Type II diabetics seem to feel better when their glycosylated hemoglobin levels are higher. Also across time, the incidence and severity of secondary complications increases significantly in both Type I and Type II diabetics (see Tables 6 and 8). In the Type I group, these physiological

changes are quite dramatic (see Table 9) and often accompanied by psychological disruption (see Table 7). Furthermore, the frequency of abnormally high levels of depression and anxiety increases dramatically in the Type I diabetics over time (see Table 10).

In the Type II group, psychological wellbeing appears to remain relatively stable and the physiological changes that occur are more gradual and less extreme than that found in the Type I group (see Tables 5 and 6).

Secondary complications are found to be associated with greater disruption in the psychological, physical, and social functioning of the individual (see Tables 15 and 16). Differences exist, however, with respect to the type and severity of secondary complication present, and the impact that it has on the physical, psychological or social functioning of the individual.

TABLE 11

CORRELATIONS: TYPE I, ONSET I AND TYPE I, ONSET II
PHYSIOLOGICAL VARIABLES AND THE SCALES ON
THE JACKSON BASIC PERSONALITY INVENTORY

VARIABLE:	HbA1C	VASCULAR	NUPATHY	NPPATHY	RTPATHY
HYP I,I	-.51	--	--	--	--
HYP I,II	.39**	.14	.41**	.26	.32
DEP I,I	-.29	--	--	--	--
DEP I,II	.32*	.05	.20	.04	.21
DEN I,I	.14	--	--	--	--
DEN I,II	.17	.12	.16	-.12	-.22
INP I,I	-.38	--	--	--	--
INP I,II	.16	-.03	.29*	.04	.29*
ALN I,I	-.02	--	--	--	--
ALN I,II	.25	-.09	-.02	.02	-.16
PID I,I	-.56	--	--	--	--
PID I,II	.28*	.00	.00	.17	.15
ANX I,I	.53	--	--	--	--
ANX I,II	.07	.04	.08	.06	.32*
THD I,I	-.46	--	--	--	--
THD I,II	.10	.13	-.03	-.01	.20
IME I,I	.29	--	--	--	--
IME I,II	.16	.12	-.04	.18	.17
SOI I,I	-.29	--	--	--	--
SOI I,II	.09	.17	.19	-.02	-.06
SDP I,I	-.14	--	--	--	--
SDP I,II	-.03	.21	.11	-.03	.06
DEV I,I	-.09	--	--	--	--
DEV I,II	.11	-.02	.06	.13	.18

* $p < .05$
** $p < .01$

TABLE 12

CORRELATIONS: TYPE II, ONSET I AND TYPE II, ONSET II PHYSIOLOGICAL VARIABLES AND THE SCALES ON JACKSON BASIC PERSONALITY INVENTORY

VARIABLE:	HbA1c	VASCULAR	NUPATHY	NPPATHY	RTPATHY
HYP II,I	-.32*	-.25	-.02	.39*	--
HYP II,II	-.23*	.11	-.03	-.02	.19
DEP II,I	-.35*	-.18	.11	-.07	--
DEP II,II	-.06	.05	.03	.27*	.01
DEN II,I	.02	.28	-.04	.03	--
DEN II,II	-.02	.15	-.02	-.00	.06
INP II,I	-.09	-.31*	-.10	.16	--
INP II,II	.17	.07	-.06	.24*	.12
ALN II,I	-.14	-.38*	-.25	-.11	--
ALN II,II	.11	-.13	.09	.09	.24*
PID II,I	-.06	-.44*	-.10	.02	--
PID II,II	-.14	.20	-.08	.12	.10
ANX II,I	-.19	-.37*	-.10	.23	--
ANX II,II	-.11	.31*	-.13	-.03	-.20
THD II,I	-.01	-.37*	-.17	.17	--
THD II,II	-.14	.12	-.19	-.22	-.09
IME II,I	-.15	-.27	.12	.38*	--
IME II,II	.18	-.11	.17	.16	.08
SOI II,I	-.17	-.23	-.08	.10	--
SOI II,II	.07	.05	.05	.12	.15
SDP II,I	-.27	-.24	.25	.05	--
SDP II,II	.07	.09	.10	.31*	.24*
DEV II,I	-.28	-.32*	.01	.05	--
DEV II,II	-.02	.05	-.01	.03	.19

* $p < .05$
** $p < .01$

TABLE 13

CORRELATIONS: TYPE I, ONSET I AND TYPE I, ONSET II
PHYSIOLOGICAL VARIABLES, METABOLIC CONTROL AND
CAMPBELL'S INDEX OF WELLBEING

		HbA1c	CONTROL	Ca9	AFFECT	WELLBEING
VARIABLE:						
VASCULAR	I,I	--	--	--	--	--
VASCULAR	I,II	.29	.02	-.07	.06	.00
NUPATHY	I,I	--	--	--	--	--
NUPATHY	I,II	.25	.29*	-.24	-.22	-.23
NPPATHY	I,I	--	--	--	--	--
NPPATHY	I,II	.42**	.30*	-.05	.02	-.01
RTPATHY	I,I	--	--	--	--	--
RTPATHY	I,II	-.09	.13	-.25	-.29	-.27
HbA1c	I,I	1.00	.41	-.93*	-.75	-.96
HbA1c	I,II	1.00	.30*	-.11	-.02	-.06
CONTROL	I,I	.41	1.00	-.52	.13	-.07
CONTROL	I,II	.30*	1.00	.02	.09	.06
Ca9	I,I	-.93*	-.52	1.00	.28	.81*
Ca9	I,II	-.11	.02	1.00	.91**	.98**
AFFECT	I,I	-.75	.13	.28	1.00	.79*
AFFECT	I,II	-.02	.09	.91**	1.00	.98**
WELLBEING	I,I	-.96*	-.07	.81*	.79*	1.00
WELLBEING	I,II	-.06	.06	.98*	.98**	1.00

* $p < .05$
** $p < .01$

TABLE 14

CORRELATIONS: TYPE II, ONSET I AND TYPE II, ONSET II PHYSIOLOGICAL VARIABLES, METABOLIC CONTROL AND CAMPBELL'S INDEX OF WELLBEING

		HbA1c	CONTROL	Ca9	AFFECT	WELLBEING
VARIABLE:						
VASCULAR	II,I	-.28	-.07	-.02	.13	.06
VASCULAR	II,II	.05	.13	-.16	-.16	-.16
NUPATHY	II,I	-.11	.28	-.27	-.11	-.21
NUPATHY	II,II	.12	.09	-.08	-.17	-.13
NPPATHY	II,I	-.00	.20	-.04	-.10	-.08
NPPATHY	II,II	.22	.05	-.16	-.18	-.17
RTPATHY	II,I	--	--	--	--	--
RTPATHY	II,II	-.10	.24*	.01	-.17	-.09
HbA1c	II,I	1.00	.13	.26	.02	.16
HbA1c	II,II	1.00	.34**	.24**	.04	.15
CONTROL	II,I	.13	1.00	-.28	-.20	-.26
CONTROL	II,II	.34**	1.00	-.06	-.09	-.10
Ca9	II,I	.26	-.28	1.00	.64**	.91**
Ca9	II,II	.24*	-.06	1.00	.74**	.94**
AFFECT	II,I	.02	-.20	.64**	1.00	.89**
AFFECT	II,II	.04	-.09	.74**	1.00	.93**
WELLBEING	II,I	.16	-.26	.91**	.89**	1.00
WELLBEING	II,II	.15	-.10	.94**	.93**	1.00

* $p < .05$
** $p < .01$

TABLE 15

CORRELATIONS: TYPE I, ONSET I AND TYPE I, ONSET II PHYSIOLOGICAL VARIABLES AND THE SCALES ON THE SICKNESS IMPACT PROFILE

VARIABLE:		HbA1c	VASCULAR	NUPATHY	NPPATHY	RTPATHY
AMBUL	I,I	--	--	--	--	--
AMBUL	I,II	.00	.10	.50**	.04	.18
ALERT	I,I	--	--	--	--	--
ALERT	I,II	.08	-.16	-.10	-.10	.28
COMMUN	I,I	--	--	--	--	--
COMMUN	I,II	.03	-.14	-.20	-.20	.25
WORK	I,I	--	--	--	--	--
WORK	I,II	.12	-.12	.04	.04	.26*
RECREAT	I,I	--	--	--	--	--
RECREAT	I,II	.01	-.15	-.05	-.04	.31*
EMOTION	I,I	--	--	--	--	--
EMOTION	I,II	.17	-.09	.09	.11	.22
MOBILITY	I,I	--	--	--	--	--
MOBILITY	I,II	-.03	-.14	.40**	-.12	.06
SOCIAL	I,I	--	--	--	--	--
SOCIAL	I,II	-.03	-.13	.16	-.01	.32*
PHYSSIP	I,I	--	--	--	--	--
PHYSSIP	I,II	-.02	-.13	.41**	-.09	.07
PSYCHSIP	I,I	--	--	--	--	--
PSYCHSIP	I,II	.01	-.14	.10	-.02	.31*
TOTSIP	I,I	--	--	--	--	--
TOTSIP	I,II	-.01	-.16	.21	-.04	.28*

* $p < .05$
** $p < .01$

TABLE 16

CORRELATIONS: TYPE II, ONSET I AND TYPE II, ONSET II PHYSIOLOGICAL VARIABLES AND THE SCALES ON THE SICKNESS IMPACT PROFILE

		HbA1c	VASCULAR	NUPATHY	NPPATHY	RTPATHY
VARIABLE:						
AMBUL	II,I	-.03	.07	-.07	.15	--
AMBUL	II,II	.10	.03	-.12	-.03	.17
ALERT	II,I	-.12	-.21	.17	.02	--
ALERT	II,II	.23*	.17	.15	.10	.12
COMMUN	II,I	-.09	-.20	-.04	-.09	--
COMMUN	II,II	.04	-.11	-.08	-.22	.16
WORK	II,I	-.28	-.01	-.10	.06	--
WORK	II,II	.06	-.18	.05	.04	.07
RECREAT	II,I	-.16	.01	-.05	-.02	--
RECREAT	II,II	.05	-.09	-.06	-.16	.13
EMOTION	II,I	-.06	-.20	.05	-.02	--
EMOTION	II,II	-.09	.02	.06	.06	.14
MOBILITY	II,I	-.06	.04	-.05	-.09	--
MOBILITY	II,II	.04	.07	.08	.08	.43**
SOCIAL	II,I	-.11	-.16	-.08	.13	--
SOCIAL	II,II	.10	.03	.05	.05	.14
PHYSSIP	II,I	-.05	.03	.12	-.03	--
PHYSSIP	II,II	-.01	.03	-.04	.02	.24*
PSYCHSIP	II,I	-.12	-.18	.07	-.13	--
PSYCHSIP	II,II	.12	.06	.09	.03	.18
TOTSIP	II,I	-.15	-.16	.08	-.09	--
TOTSIP	II,II	.06	-.01	.01	-.02	.19

* $p < .05$
** $p < .01$

TABLE 17

CORRELATIONS: TYPE I, ONSET I AND TYPE I ONSET II SECONDARY COMPLIATIONS

VARIABLE:		VASCULAR	NUPATHY	NPPATHY	RTPATHY
VASCULAR	I,I	1.00	--	--	--
VASCULAR	I,II	1.00	.42**	.35*	.10
NUPATHY	I,I	--	1.00	--	--
NUPATHY	I,II	.42**	1.00	.40**	.27
NPPATHY	I,I	--	--	1.00	--
NPPATHY	I,II	.35*	.40**	1.00	.40**
RTPATHY	I,I	--	--	--	1.00
RTPATHY	I,II	.10	.27	.40**	1.00

TABLE 18

CORRELATIONS: TYPE II, ONSET I AND TYPE II, ONSET II SECONDARY COMPLIATIONS

VARIABLE:		VASCULAR	NUPATHY	NPPATHY	RTPATHY
VASCULAR	II,I	1.00	.17	-.18	--
VASCULAR	II,II	1.00	.21	.22	.09
NUPATHY	II,I	.17	1.00	-.05	--
NUPATHY	II,II	.21	1.00	1.00	.43**
NPPATHY	II,I	-.18	-.05	1.00	--
NPPATHY	II,II	.22	.39**	1.00	.19
RTPATHY	II,I	--	--	--	1.00
RTPATHY	II,II	-.16	-.08	-.16	1.00

* $p < .05$
** $p < .01$

TABLE 19

CORRELATIONS: TYPE I, ONSET I AND TYPE I, ONSET II METABOLIC CONTROL, CAMPBELL'S INDEX OF WELLBEING AND SCALES ON THE SICKNESS IMPACT PROFILE

VARIABLE:		HbA1c CONTROL	Ca9 AFFECT	WELLBEING		
AMBUL	I,I	N/A	-.25	.11	-.29	-.13
AMBUL	I,II	.00	.20	-.12	-.18	-.15
ALERT	I,I	-.20	.42	.11	.52	.39
ALERT	I,II	.08	-.05	-.58**	-.64**	-.62**
COMMUN	I,I	.29	.58	.11	.39	.39
COMMUN	I,II	.03	-.08	-.53**	-.54**	-.55**
WORK	I,I	-.30	.42	.06	.57	.38
WORK	I,II	.12	.15	-.54**	-.47**	-.51**
RECREAT	I,I	.00	.06	.07	.08	.10
RECREAT	I,II	.01	-.03	-.60**	-.65**	-.63**
EMOTION	I,I	-.10	.41	.14	.43	.35
EMOTION	I,II	.17	.19	-.67**	-.60	-.65**
MOBILITY	I,I	-.29	.38	.11	.54	.39
MOBILITY	I,II	-.03	-.07	-.24	-.35*	-.30*
SOCIAL	I,I	-.23	-.53	-.50	-.57	-.71
SOCIAL	I,II	-.03	-.08	-.73**	-.72**	-.74**
PHYSSIP	I,I	-.29	.28	.15	.42	.33
PHYSSIP	I,II	-.02	-.04	-.18	-.26	-.22
PSYCHSIP	I,I	-.15	.32	-.13	.17	.01
PSYCHSIP	I,II	.01	-.04	-.74**	-.74**	-.75**
TOTSIP	I,I	-.19	.32	-.03	.28	.14
TOTSIP	I,II	-.01	-.03	-.67**	-.69**	-.69**

* $p < .05$
** $p < .01$

TABLE 20

CORRELATIONS: TYPE I, ONSET I AND TYPE I, ONSET II
METABOLIC CONTROL, CAMPBELL'S INDEX OF WELLBEING
AND SCALES ON THE SICKNESS IMPACT PROFILE

		HbA1c	CONTROL	Ca9	AFFECT	WELLBEING
VARIABLE:						
AMBUL	II,I	-.03	.08	-.04	.04	-.01
AMBUL	II,II	.10	-.06	-.16	-.21	-.19
ALERT	II,I	-.12	.17	-.01	-.37*	-.20
ALERT	II,II	.23*	.17	-.28	-.41**	-.37**
COMMUN	II,I	-.09	.14	-.15	-.48**	-.34*
COMMUN	II,II	.04	.08	-.02	-.19	-.11
WORK	II,I	-.28	-.03	.02	-.35*	-.18
WORK	II,II	.06	.23*	-.13	-.14	-.15
RECREAT	II,I	-.16	.14	.01	-.32*	-.17
RECREAT	II,II	.06	.04	.24*	.12	.20
EMOTION	II,I	-.06	.19	.02	-.54**	-.28
EMOTION	II,II	-.09	.10	-.32**	-.43**	-.40**
MOBILITY	II,I	-.06	.27	-.37*	-.30*	-.37*
MOBILITY	II,II	.04	.15	-.17	-.38**	-.29*
SOCIAL	II,I	-.11	.13	-.05	-.59**	-.35*
SOCIAL	II,II	.10	.06	-.10	-.25*	-.19
PHYSSIP	II,I	-.05	.23	-.27	-.21	-.27
PHYSSIP	II,II	-.01	-.02	-.22*	-.29*	-.27*
PSYCHSIP	II,I	-.12	.10	-.03	-.55**	-.31*
PSYCHSIP	II,II	.12	.14	-.24*	-.41**	-.35**
TOTSIP	II,I	-.15	.14	-.11	-.57**	-.37*
TOTSIP	II,II	.06	.07	-.24*	-.38**	-.33**

* $p < .05$
** $p < .01$

2. What differences, if any, are there in the psychological functioning of persons who have recently been diagnosed with diabetes in comparison with persons who have lived with diabetes for several years?

According to the results of our study, psychological differences between recently diagnosed and long duration diabetics are much more significant with the Type I group in comparison to the Type II group. Findings reveal that Type I diabetics undergo dramatic detrimental psychological changes across time (see Tables 7 and 10). Over time, Type I diabetics are found to score significantly higher on several psychological scales of the Jackson Basic Personality Inventory, for example, Depression, Persecutory Ideas, Self-depreciation and Deviation scales. In addition, the frequency of pathological levels of anxiety and depression is much higher in long duration Type I diabetics in comparison to the other three diabetic groups. From a 0% incidence of severe depression and anxiety in recently diagnosed diabetics, Type I diabetics shift to a 22% incidence of severe depression and a 16% incidence of severe anxiety over time. Therefore, over time the Type I diabetics are likely to experience increasing difficulty with social, emotional and psychological functioning.

By comparison, the Type II group appears to remain more psychologically stable across the lifespan. With

respect to the twelve scales on the Jackson Basic Personality Inventory, no overall significant differences in scores were found to occur over time in the Type II group. (see Table 5). Furthermore, the frequency rates of severe anxiety and depression were found to be more stable across time for the Type II diabetics. The incidence of severe depression was found to change by only 2% across time, and the incidence of severe anxiety increased only by 3% over time.

In summary, it appears that differences in the psychological functioning that occur over time in diabetics are strongly influenced by the diabetes Type. Psychological changes which occur in the Type I diabetics are quite dramatic in comparison to the relatively stable Type II diabetics.

3. Do diabetics seem to adapt to their condition over time or do adverse psychological effects of the disease increase over time?

According to the data presented in Table 5, Type II diabetics seem to adapt quite well psychologically. Overall, there is no significant difference between recently diagnosed and long duration Type II diabetics with respect to scores obtained on the twelve psychological scales of the Jackson Basic Personality Inventory. Despite significant physiological changes in these individuals (see Table 6), they remain relatively unaltered psychologically.

The only significant psychological change that occurs with these individuals is with respect to more physical complaints and health concerns. Such complaints are reasonable in view of the secondary complications which develop, and the normal physical changes that frequently accompany the aging process.

In comparison to the Type II diabetics, the Type I diabetics seem to have greater difficulty adapting to their illness. In general, it appears that the psychological difficulties that the Type I diabetics experience are related to the dramatic physical changes that occur over time with this group. In our recently diagnosed Type I group, no signs of severe anxiety, severe depression, or secondary complications were present (see tables 9 and 10). Overall, our recently diagnosed Type I diabetics presented as a physically and psychologically healthy group. However across time, Type I diabetics were found to develop serious psychological and physical disturbances (see Tables 7 - 10). It appears that the dramatic physiological changes that sometimes occurs in these individuals seriously challenges their adaptive capacities, and in many cases, the outcome is psychological disruption reaching pathological levels.

4. What impact does the severity of the disease, secondary complications, and metabolic control have on the psychological and social functioning of diabetics?

In Type I diabetics, poorer metabolic control is

found to be significantly correlated with lower scores on Campbell's Life Satisfaction, and Wellbeing scales (see Table 13). In addition, higher glycosylated hemoglobin levels are found to be significantly correlated with higher scores on the Hypochondriasis, Depression, and Persecutory Ideas scales of the Jackson Basic Personality Inventory (see Table 11). These results suggest that Type I diabetics in poor metabolic control are more likely to feel threatened, depressed, and generally unhappy and dissatified with their lives. Furthermore, they are more likely to feel worse physically, and may frequently complain about physical symptoms or disturbances.

In Type I diabetics, secondary complications such as retinopathy and neuropathy are found to be significantly related to higher scores on the Hypochondriasis and Interpersonal Problems scales of the Jackson Basic Personality Inventory. In addition, retinopathy is found to be significantly related to higher levels of Anxiety (see Table 11), and disrupted work, recreational, social, psychosocial, and total life functioning (Sickness Impact Profile) (see Table 15). Lastly, neuropathy in Type I diabetics is found to be positively correlated with disturbances in ambulation, mobility, and physical functioning (Sickness Impact Profile) (see Table 15). These results illustrate the

detrimental impact of secondary complications, such as retinopathy and neuropathy, on the diabetics's physical, psychological, and social life functioning. In summary, it appears that high glycoyslated hemoglobin levels and/or the appearance of secondary complications in Type I diabetics can seriously alter the physical, social and psychological functioning of the affected individual. Such individuals are much less likely to find life enjoyable.

With the Type II group, higher glycosylated hemoglobin levels, and secondary complications such as nephropathy and neuropathy are found to be significantly associated with higher scores on the Self-depreciation scale of the Jackson Basic Personality Inventory (see Table 12). It appears that Type II who have difficulty controlling their glycosylated hemoglobin levels, and/or who develop physical complications as a result of their illness, may feel worthless, inadequate, or incapable of managing their own disease condition. In addition, increased severity of physical complications is shown to be significantly correlated with fncreased levels of Depression or Interpersonal Problems (nephropathy), and an increased tendency toward feelings of Alienation (retinopathy) (see Table 12). Furthermore, according to the scores on the Sickness Impact Profile, Type II diabetics with retinopathy are shown to experience significant

difficulties with mobility and physical functioning (see Table 16).

In summary, it appears that the risk of developing disturbances of a physical, psychological and/or social nature is increased in diabetics who demonstrate high glycosylated hemoglobin levels, and/or who show evidence of the development of secondary complications. Furthermore, the detrimental influence of these variables appears to be more evident with the Type I diabetics in comparison to the Type II diabetics.

5. What impact does physical disability have on the psychological and social well-being of diabetics?

The presence of secondary complications places restrictions on the diabetic's lifestyle which interferes with the individual's physical and social functioning. This in turn, has negative psychological consequences. In long duration diabetics, secondary complications such as neuropathy, nephropathy, vascularity, and retinopathy are associated with higher levels of Depression, Anxiety, Interpersonal problems, Self-depreciation, and feelings of Alienation (see Tables 11 and 12). In addition, where there is disruption with mobility, general wellbeing and life satisfaction is reduced (Tables 19 and 20).

Perhaps the most difficult secondary complication that a diabetic may have to adjust to is retinopathy.

Retinopathy is shown to have the potential to seriously disrupt several aspects of the diabetic's life. Mobility is severely reduced with retinopathy. In addition, the mental alertness of the diabetics with retinopathy may be affected, disrupting communication, social and recreational aspects of their lives (see Table 16).

In summary, the results of our study indicate that whenever physical disability arises, the likelihood of disturbances in social and/or psychological functioning is also increased.

6. Does metabolic control decline or improve across time and what is the relationship between metabolic control and psychological disorder?

In both Type I and Type II diabetics, there is an overall increase in the glycosylated hemoglobin level between the recently diagnosed and long duration diabetics (see Table 2). With the Type II diabetics, these changes in the glycosylated hemoglobin levels across time are statistically significant (see Table 6). Glycosylated hemoglobin levels seems to increase significantly across the life span in Type II diabetics. The same is not true of Type I diabetics (see Table 8).

According to Table 11, higher glycosylated hemoglobin levels in Type I diabetics are significantly related to higher scores on the Hypochondriasis, Depression, and Persecutory Ideas scale of the Jackson Basic Personality

Inventory. In addition, higher glycosylated hemoglobin levels are negatively associated with feelings of general wellbeing. These results illustrate how important it is for Type I diabetics to adhere to their diabetic regimens in order to maintain good metabolic control. Type I diabetics maintaining normal glycosylated hemoglobin levels are much more likely to feel better both physically and psychologically. Consequently, they will have a more positive outlook about their future, about themselves, and about life in general.

Although Type II diabetics are found to show increases in glycosylated hemoglobin levels across the lifespan, higher glycosylated hemoglobin levels in Type II diabetics are found to be associated with lower scores on the Hypochondriasis and Depression scales of the Jackson Basic Personality Inventory (see Table 12), with higher levels of Alertness (see Table 20), and with greater life satisfaction (see Table 14). These results are consistent with research that has reported diabetics perceiving increased feelings of alertness and general wellbeing with higher glycosylated hemoglobin levels.

7. What is the relationship between psychopathology and diabetes mellitus across time, and does the incidence of psychopathology appear to be related to metabolic control or secondary complications?

According to results shown in Table 10, the likelihood

of finding abnormally high levels of depression and/or anxiety in diabetics seems to increase across the lifespan. The change in Type I diabetics is most pronounced with 22% of the long duration diabetics exhibiting severe levels of depression, and 16% exhibiting severe levels of anxiety. This is in comparison to a 0% incidence of severe depression or anxiety being found in the recently diagnosed Type I group. With the Type II group, frequency rates of severe depression and anxiety are less evident, and changes occurring across time, less dramatic than the Type I group. The rate of severe depression is even shown to decline somewhat across time in Type II diabetics from 9% in the recently diagnosed group, to 7% in the long duration Type II group. In the case of severe anxiety, rates are found to increase slightly over time from 6% in the recently diagnosed to 9% in the long duration Type II group. Therefore, it does appear that the incidence of psychopathological levels of anxiety and depression increase across time. These changes are most notable in the Type I diabetic group.

Secondary complications are also shown to increase in rate and severity across time (see Table 9). Neuropathy and nephropathy are found to increase in rate and severity significantly over time in the Type II group (see Table 6), and retinopathy is found to increase in rate and severity

significantly over time in the Type I group (see Table 8). Furthermore, glycosylated hemoglobin levels are shown to increase in both the Type I and Type II across time. With the Type II group these changes are statistically significant (see Tables 6 and 8).

Correlational tables show a strong positive relationship between severity of secondary complications and scores on several psychological scales of the Jackson Basic Personality Inventory (see Tables 11 and 12). Neuropathy is found to be significantly correlated with Hypochondriasis and Interpersonal Problems (in Type I diabetics); nephropathy is found to be significantly correlated with Hypochondriasis, Depression, Interpersonal Problems, Impulse Expression, and Self-depreciation (in Type II diabetics); vascularity is found to be significantly correlated with Anxiety (Type II diabetics); and retinopathy is found to be significantly correlated with Hypochondriasis, Anxiety, and Interpersonal Problems (in Type I diabetics), and with Alienation and Self-depreciation in Type II diabetics.

Lastly, higher glycosylated hemoglobin levels, representing poorer metabolic control, are shown to be significantly correlated with increasing severity of neuropathy, vascularity, and nephropathy in the Type I group, and with an increasing rate of retinopathy in the

Type II group (see Tables 13 and 14).

In summary, it appears that the incidence of psychological disturbance or psychopathology is related to the incidence and severity of secondary complications. Both the frequency of secondary complications and psychopathology increase correspondingly across the diabetic lifespan. In addition, there is evidence that high glycosylated hemoglobin levels may increase the risk of the developing secondary complications and/or may hinder the individual's ability to enjoy life and feel well.

8. What similarities or differences exist between Type I and Type II diabetics with respect to psychological, social or physical functioning?

According to the results presented in Tables 9 and 10, the frequency of severe complications and pathological levels of depression and anxiety are higher in the long duration Type I group in comparison to Type II diabetics. While Type I diabetics initially appear to fair better both physically and psychologically after recent diagnosis, the changes that occur over time in the Type I group are more dramatic and detrimental. Psychologically, Type II diabetics do not change significantly across time as indicated in Table 5. The only exception is a higher rate of physical complaints as indicated by increasing scores on the Hypochondriasis scale of the Jackson Basic Personality Inventory. Type I diabetics are shown to change over time

with respect to several psychological variables (see Table 7). Scores on the Depression, Persecutory Ideas, Self-depreciation, and Deviation scales of the Jackson Basic Personality Inventory are found to be significantly higher in the long duration Type I group in comparison to recently diagnosed Type I diabetics. Therefore, across time the Type I diabetics seem to undergo greater psychological disruption than the Type II diabetics.

Physically, the Type I diabetics also seem to encounter some dramatic changes over time. Secondary complications of neuropathy, nephropathy, vascularity, and retinopathy become evident in long duration diabetics, whereas none was found in the recently diagnosed Type I group (see Table 10). In Type II diabetics, secondary complications are evident in both the recently diagnosed and long duration diabetics. Long duration Type I and Type II diabetics are very similar with respect to the incidence of secondary complications (70% and 71% respectively), and similar in terms of the incidence of 'severe' secondary complications (35% and 30% respectively). Where these two groups differ notably is with regards to the rate and extent of physical complications, and in regards to the incidence of diabetic retinopathy. 58% of the long duration Type I diabetics show signs of retinopathy in comparison to 15% of the long duration Type II diabetics. An examination of Tables 15 and 16, reveals that

retinopathy and other secondary complications may significantly disrupt mobility, social functioning, physical functioning, and the total life functioning of the diabetic individual. Furthermore, the development of one secondary complication is often accompanied by the development of one or more secondary complications (see Tables 17 and 18). Retinopathy is significantly correlated with neuropathy and nephropathy; neuropathy is significantly correlated with nephropathy; and vascularity is significantly correlated with vascularity and neuropathy.

In conclusion, long duration Type I and Type II diabetics share many similarities such as increased rates of secondary complications, increased rates of severe anxiety (see Table 10), and associated disruptions in physical and social functioning when compared with the corresponding recently diagnosed group. Where the Type I and Type II diabetics differ is with respect to the physical and psychological changes that occur over time. Type I diabetics change significantly both physically and psychologically after initial diagnosis. Furthermore, the increased incidence of retinopathy in long duration Type I diabetics makes this group more likely than Type II diabetics to experience significant difficulties with social and psychosocial functioning than Type II diabetics.

V. DISCUSSION

Major Findings

The purpose of this study was to investigate the psychological impact of Type I and Type II diabetes on adult diabetics over time. In order to do this, comparisons were made between recently diagnosed and long duration Type I and Type II diabetics, and between Type I and Type II diabetics. Results of this study reveal several interesting findings in terms of the psychological and physiological changes that occur across time in both Type I and Type II diabetics. Following is a brief summary of the major findings of this study as interpreted by this investigator:

1. There are distinct differences between Type I and Type II diabetics in terms of the physiological changes that occur over time (ie. HbAlc levels), in terms of the secondary complications that they are more likely to develop, and in terms of the psychological changes that occur over time.
2. In Type I diabetics, significant psychological and physiological changes occur across time. In Type II diabetics, significant physiological, but not psychological, changes occur across time.
3. The physiological and psychological changes that occur over time in Type I diabetics are much more severe than

that found to occur in Type II diabetics.

4. Rates of pathological anxiety and depression are found to be noteably higher in Type I diabetics than Type II diabetics.

5. The development of secondary complications with corresponding physical disability appears be associated with psychological disruption. Retinopathy is shown to be particularly disrupting to total life functioning.

6. Type I diabetics tend to have glycosylated hemoglobin levels which are, on the average, higher than Type II diabetics.

7. Glycosylated hemoglobin levels increase significantly across time in Type II diabetics, but not Type I diabetics.

8. Type I diabetics appear to feel better when their glycosylated hemoglobin levels are lower; Type II diabetics appear to feel better when their glycosylated hemoglobin levels are higher.

9. Glycosylated hemoglobin levels appear to be associated with the development of certain secondary complications. However, differences exist according to diabetic type.

Physiological Changes

Across the diabetic lifespan, adverse physiological changes are noted to occur in both the Type I and Type II diabetics. Glycosylated hemoglobin levels increase over time (significantly in Type II diabetics), and rates and

severity of secondary complications also increase across the diabetic lifespan. The increase in rates of physical complications across time for the Type I group were notably high (70%), and rates of 'severe' secondary complications increased 35% after ten or more diabetic years. By comparison, the Type II group showed a 21% increase in secondary complications over time, and a 22% increase in 'severe' secondary complications. Therefore, our findings are consistent with research that has reported a significant relationship between the development of secondary complications and longer diabetic duration. (Barglow, Hatcher, Edidin, 1984; Clements & Bell, 1985; Bradley, 1982; Godine, 1988; Klein, Moss & Klein, 1987; Teuscher, Schnell & Wilson, 1988). However, the rates of change across time were much higher in our Type I group in comparison to our Type II group.

In contrast to the findings of several research studies, (Bild, et al., 1989; Clements & Bell, 1985; Godine, 1988), our study failed to clearly establish a significant relationship between hyperglycemia/poor metabolic control and the development of secondary complications in all cases. Increases in the rates and severity of secondary complications in our Type I group were higher than that in our Type II group despite no significant change in glycosylated hemoglobin levels in our

Type I group. Nevertheless, correlational analysis did reveal a significant relationship between poorer metabolic control and neuropathy in Type I diabetics; between poorer metabolic control and nephropathy in Type I diabetics; and between poorer metabolic control and retinopathy in Type II diabetics. In addition, high glycosylated hemoglobin levels were found to be significantly related to the development of nephropathy in Type I diabetics. Therefore, our study finds the development of secondary complications to be associated with diabetic type and duration, moreso than metabolic control. However, our study examined glycosylated hemoglobin levels only at a single point across the diabetic lifespan. Consequently, we do not know how well our diabetic subjects maintained metabolic control over a period of months or years. Similarly, our study did not examine the number of serious episodes of hyperglycemia or hypoglycemia that our diabetics may have experienced. Such episodes may have an influence upon the development of chronic secondary complications. In addition, Godine, (1988), has suggested other factors which may influence the development of secondary complications to include genetic predisposition, hypertension, and environmental factors.

As is apparent, our study found distinct differences between Type I and Type II diabetics with respect to the types of secondary complications that they were likely to

develop. Rates of retinopathy were found to be much higher in Type I diabetics compared to Type II diabetics. This was expected. Retinopathy is normally reported as being much more common in IDDM diabetics compared to NIDDM diabetics. According to the Clements & Bell, (1985), it is anticipated that 60% of IDDM patients will show signs of diabetic retinopathy and 50% of NIDDM diabetics will show signs of retinopathy after ten or more diabetic years. Our results showed a comparable rate of diabetic retinopathy in Type I diabetics (50%) but a much lower than expected rate in our Type II group (18%). As the glycosylated hemoglobin levels in our subjects were generally found to be lower than that normally reported in other studies, it is possible good metabolic control may have contributed to the lower than expected rate of retinopathy in our Type II group.

Results of our study also revealed that rates of retinopathy increased significantly over time in our Type I diabetics, but not Type II, diabetics. None of our recently diagnosed Type I diabetics presented with signs of retinopathy whereas half of our long duration Type I diabetics did. This also was not unexpected as the development of retinopathy is usually not observed in Type I diabetics until after five or more years diabetic duration. Thereafter, the prevalence rate of retinopathy is known to increase dramatically, reaching 80%

after 30 years of diabetes (Clements & Bell, 1985).

In our Type II group, rates and severity of nephropathy, neuropathy, and vascularity were found to be much higher than that in our Type I group. Yet, these complications are normally found to be more common in Type I than Type II diabetics (Clements & Bell, 1985; Bild, et al., 1989). Our study found the reverse, with 15% of Type I, and 23% of Type II diabetics demonstrating symptoms of nephropathy; 37% of Type I diabetics and 52% of Type II diabetics demonstrating symptoms of neuropathy; and, 5% of Type I and 39% of Type II diabetics demonstrating symptoms of vascularity.

Development of neuropathy and nephropathy were shown to increase significantly over time in our Type II, but not Type I group. Neuropathy was shown to increase almost double, from 33% in recently diagnosed Type II diabetics, to 64% in long duration Type II diabetics. However, cited in the literature are reports that the symptoms of neuropathy are usually not present at the time of diagnosis, (Clements & Bell, 1985), and yet individuals in both our recently diagnosed Type I and Type II groups presented with such symptoms.

Psychological Changes

Results revealed distinct differences between Type I and Type II diabetics in terms of the psychological changes

that occur over time. Again, the degree and extent of psychological changes were more dramatic and apparent in the Type I group when compared with the Type II group. It has been widely reported that the initial diagnosis of diabetes is a very traumatic experience for the individual and his or her family (Fujita, Kobayashi & Imamura, 1967; Levin, et al., 1986). Upon learning of the diagnosis, common responses of the diabetic individual include feelings of anger, anxiety, guilt, depression, and denial (Citrin, et al., 1986). In accordance with these findings, there was evidence to suggest that some of our recently diagnosed Type II diabetics might be encountering psychological difficulty with either adapting or adjusting to their diabetes. This was indicated from the relatively high scores recently diagnosed Type II diabetics measured on scales of denial, alienation, persecutory ideas, and thinking disorder. Pathological levels of anxiety and depression were also evident in some of the members belonging to this group. In contrast to this, our recently diagnosed Type I diabetics presented as being atypical in terms of psychological adjustment and well-being. They tended to score relatively high on measures of life satisfaction, positive affect, self efficacy and general wellbeing, and relatively low on measures of depression, anxiety or denial. In addition, pathological levels of

anxiety or depression were not found in this group. This very small group of Type I individuals seemed to possess extremely good coping skills and adaptive capacity. Results suggested our recently diagnosed Type I diabetics were quite happy and content with their lives. They seemed to be able to maintain a positive outlook on life despite their recent diabetic diagnosis. In addition, their relatively low scores on the denial scale suggests that their well-adjusted psychological state was not likely an overly-optimistic attitude as these individuals were probably not using denial as a coping strategy. Nevertheless, it is likely that many recently diagnosed Type I diabetics (not included in this study) do experience great psychological distress in relation to their diagnosis and recent adjustment to diabetes. However, they may not have wished, nor have been psychologically able to participate in the diabetic outpatient program.

Across time, significant psychological changes were noted to occur in the Type I, but not Type II, diabetics. Whereas Type II diabetics tended to remain relatively psychologically stable, some of our long duration Type I diabetics showed signs of severe psychological disruption. There were significant increases on scales measuring depression, persecutory ideas, self-depreciation and deviation; pathological levels of depression reached 22%

and pathological levels of anxiety reached 16% in long duration Type I diabetics. Clearly, many long duration Type I diabetics were experiencing serious psychological discomfort. Several studies have indicated a relationship between diabetes and psychiatric illness. Lustman, et al., (1986), found that of the 114 diabetic patients they studied, 71% of them had a history of at least one defined psychiatric disorder. Similarly, Swift, Seidman & Stein (1967), found psychiatric illness to be much more commonly found in diabetics than nondiabetics, and Lustman, Griffith and Clouse (1988), concluded from their study of diabetics, that more than 50% of all diabetics experience some form of psychiatric illness at some time during their lives. In particular, anxiety and depression are two of the most commonly cited psychiatric problems which have been found to accompany diabetes. Prevalance figures for current depression are believed to approximate 20-35% depending upon the criteria used to diagnose depression. Lustman, et al., (1986), found 33% of the 114 diabetic patients he studied received a major diagnosis of depression and, Popkin, et al. (1988), found 24% of the 75 long duration Type I diabetics they studied received a diagnosis of major depression. Our study found rates of pathological depression to range from 0-22% depending upon diabetic type and duration. In addition, Lustman, et al., (1986),

reported 41% of their diabetic subjects received a diagnosis of generalized anxiety disorder, whereas Popkin, et al., (1988), reported a 31.7% prevalence rate of anxiety disorder in the diabetics they studied. Our study found rates of pathological anxiety to range from 0-16% depending upon diabetic type and duration. However, Lustman (1986) reported no significant difference between Type I and Type II diabetics in terms of the overall prevalence rate of psychiatric disorder, and Popkin, et al., (1988), found that prevalence rates for major depression were unrelated to diabetic duration. Our findings were similar to Popkin, et al, (1988), in that rates of severe depression did not appear to increase significantly over time in our Type II diabetics. However, in a follow up conducted by Lustman, Griffith & Clouse (1988), it was found that rates of depression did increase substantially over time. After a five year period, 64% of the diabetics they examined reported experiencing at least one episode of depression since diagnosis. In addition, results from our study indicated that over time our Type II diabetics complained more about physical symptoms and bodily ailments as revealed by their higher scores on the Hypochondriasis scale. This is not surprising given the declining physical status and older age of many of our long duration Type II group. A greater number of somatic complaints (in addition

to anxiety and depression), has often been found to accompany the onset of physical complications (Holmes, D., 1986). The increasing number of physical complaints expressed by such diabetics likely accounts for the larger number of prescription drugs also taken by diabetics in comparison to nondiabetics. Such prescriptions include medications for illnesses other than diabetes (Isacon & Stalhammer, 1987).

The Relationship Betweeen Physiological and Psychological Functioning

The overall findings of this study indicate that the more that the physical functioning of the individual is affected by diabetes, the more likely they are to experience serious psychological disruption. These findings are consistent with research which has indicated significant relationships between overall physical health, functional ability and subjective wellbeing (Speitzer, Snyder & Larson, 1979-1980; Felton, Reveson & Hinrichsen, 1984); between loss of function and chronic emotional stress (Davidson, 1981); and between physical illness and depression (Lustman, 1988). Increased rates and severity of physical complications in our diabetic subjects, were found to be accompanied by increased levels of anxiety and depression, and higher scores on measures of self-depreciation, interpersonal problems, alienation and

hypochondriasis. It is believed that the drastic increase in rates of severe anxiety and depression in our Type I diabetics was largely related to the marked increase in rates of retinopathy in these diabetics. Retinopathy is commonly found to have a devastating impact upon the diabetic individual's life (Wulsin, Jacobson & Rand, 1987). Our study revealed retinopathy as significantly altering three broad aspects of the individual's life: their physical functioning, their social functioning, and their psychological functioning. An analysis of the correlations conducted revealed that retinopathy significantly disrupted the work, mobility, recreation, and social interaction of our diabetics with retinopathy. In addition, retinopathy was found to be significantly associated with a greater number of interpersonal problems, lower levels of self-worthiness and self-esteem, and a greater tendency towards feeling alienated from others. Similarly, Oehler's (1978), cross sectional study of 33 diabetic retinopaths found retinopathy to limit not only the individual's physical activity, but the individual's social, financial, and leisure activities as well, and Jacobson and Leibovich (1984), found that the onset of retinopathy increased psychological distress not only because of the changing vision, but because of other negatively experienced life events that were associated with the adjustment to

retinopathy. However, retinopathy was not the only physical complication found to negatively affect the psychological well-being of the diabetic. Neuropathy was found to be significantly associated with interpersonal problems in Type I diabetics; nephropathy was found to be significantly associated with depression, interpersonal problems and self depreciation in Type II diabetics; and vascular symptoms were found to be significantly associated with anxiety in Type II diabetics. Therefore, results of our study are consistent with research which has found a significant relationship between the development of secondary complications and increased levels of fear, anxiety, depression, withdrawal, physical compaints, guilt, anger, and self-blame (Davidson, 1981; and Holmes, D., 1986).

Several studies have reported an association between metabolic control and psychological functioning, and between hyperglycemia and psychiatric disorder. However, the findings of these studies have often been contradictory. In many cases, results appear to differ according to degree of metabolic control and diabetic type. In addition, the subjective symptoms of abnormal blood glucose levels have consistently been found to differ greatly from person to person (Gonder-Frederick, et al., 1989). Some researchers have reported good metabolic

control to be associated with better mental health (Tsuang, Perkins & Simpson, 1983), lower levels of anxiety and depression, better quality of life (Rodin, 1983; Mazze, Lucido & Shamooon, 1984), and a lower prevalence of psychiatric disorder (Lustman, 1988). In contrast, other studies have reported lower blood glucose levels to be associated with greater anxiety and irritability, and higher blood glucose levels to be associated with greater mental alertness and positive affect (Gonder-Frederick, et al., 1989). Our study finds differences between Type I and Type II diabetics with respect to the influence of glycosylated hemoglobin levels on psychological wellbeing. With our Type I diabetics, poorer metabolic control as indicated by higher glycosylated hemoglobin levels, were found to be significantly related with higher levels of depression, and lower levels of life satisfaction and general wellbeing. Similarly, the number of physical complaints and persecutory ideas expressed by these individuals was also shown to increase when metabolic control was poor. However, our Type II diabetics presented with a reverse relationship. In these diabetics, higher glycosylated hemoglobin levels were found to be associated with lower levels of depression, less physical complaints, and greater satisfaction in life. The latter findings are consistent with the results of a study which reported

diabetics felt subjectively better when glycosylated hemoglobin levels were moderately high (HbA1 < 10). Very few of our Type II diabetics presented with glycosylated hemoglobin levels above 10. Consequently, when attempting the establish the relationship between elevated glycosylated hemoglobin levels and psychological wellbeing, it is necessary to consider the degree of metabolic control or hyperglycemia.

In addition, some researchers believe that the impact of hyper or hypoglycemia on Type I diabetics may be more harmful than that in Type II diabetics. Surridge, et al., (1984), found that psychiatric disorder was more prevalent in IDDM patients who had very high glycosylated hemoglobin levels. It has also been suggested that hyperglycemia seriously affects the central nervous system in Type I diabetics. This damage is believed to often lead to neuropsychological and psychiatric changes (Ryan, et al., 1984: Rovet, Ehlrich & Hoppe, 1988; Ryan & Morrow; 1986). However, Wilkinson, et al., (1987), found psychiatric disorder to be unrelated to glycosylated hemoglobin levels in the 194 IDDM diabetics that he studied.

In conclusion, the relationship between metabolic control and psychological functioning in diabetics remains somewhat unclear. Nevertheless, results of our study

reveal differences in the influence of glycosylated hemoglobin levels on Type I and Type II diabetics.

Summary of Discussion

Type I Diabetics

Type I diabetics undergo psychological changes across time. The decline in physical health that these diabetics undergo as a result of their illness is shown to be accompanied by a substantial decline in their psychological well-being. In particular, the rise in the number of Type I diabetics demonstrating symptoms of retinopathy is believed to be a major factor contributing to the higher rates of pathological depression and anxiety in these diabetics. Retinopathy was shown to affect the total life functioning of the individual, and the accompanying psychological disruption appeared to stem from the physical restrictions that naturally occur as a result of visual impairment. However, retinopathy was not found to be significantly related to glycosylated hemoglobin levels in Type I diabetics. Glycosylated hemoglobin levels were shown to remain relatively stable across time in Type I diabetics, while the increase in retinopathy was statistically significant. Therefore, the relationship between elevated glycosylated hemoglobin levels and the development of retinopathy was not found to be significant in this study. Rather, poor metabolic control was found to

be significantly correlated with the development of neuropathy and nephropathy in Type I diabetics. In addition, elevated glycosylated hemoglobin levels were found to have a negative impact upon the psychological well-being of Type I diabetics.

Type II Diabetics

Type II diabetics undergo significant physical, but not psychological, changes across time. Indeed, many of the recently diagnosed Type II diabetics were already experiencing symptoms of secondary complications. Rates of neuropathy and nephropathy were found to increase significantly across time in Type II diabetics and was accompanied by a significant increase in glycosylated hemoglobin levels. However, correlational analysis indicated that elevated glycosylated hemoglobin levels were not significantly associated with the development of these secondary complications. Only retinopathy was found to be significantly related with higher glycosylated hemoglobin levels in our Type II diabetics. Lastly, higher glycosylated hemoglobin levels were found to be associated with greater psychological well-being and life satisfaction in Type II diabetics.

Measures

Our study found that the following measures were useful in providing good comprehensive information with

regards to the psychological impact of diabetes from the diabetics' perspective: (1) The Jackson Basic Personality Inventory - particularly, the Hypochondriasis, Depression, Anxiety, Self-Depreciation, Interpersonal Problems and Persecutory Ideas Scales. (2) Campbell's Scale of Wellbeing - all three scales and (3) the Sickness Impact Profile - particularly, the Psychosocial Sickness Impact Profile, the Total Sickness Impact Profile, and the following subscales: Work, Alertness, Emotional Behavior, Communication, Social Interaction, Recreation and Mobility.

Where Our Findings Differed

In contrast to much research which has pointed to an association between elevated glycosylated hemoglobin levels and secondary complications, our study revealed that the development of secondary complications was largely determined by diabetic type, diabetic duration, and diabetic control (ie. glycosylated hemoglobin levels). Retinopathy was found more common in Type I diabetics regardless of glycosylated hemoglobin levels, and symptoms of neuropathy, nephropathy and vascular problems were more common in Type II diabetics independent of degree of metabolic control. Also, in contradiction to much research which has indicated an association between metabolic control and psychological wellbeing, our study revealed hyperglycemia as having both a positive and a

negative impact upon the diabetic's subjective wellbeing. Differences in terms of how the psychological state of the individual was affected by poor metabolic control was largely dependent upon the type of diabetes the individual had. Furthermore, we found that most of our diabetics adapted quite well to their illness despite much literature which would lead us to expect high levels of adjustment problems. When serious psychological difficulties were noted in our diabetics, these appeared to be related to the development and degree to which secondary complications imposed restrictions upon their lifestyle. Finally, our study showed that there were distinct differences between Type I and Type II diabetics in terms of the physiological and psychological changes that occurred over time. Type I diabetics appeared to undergo both extreme physical and psychological changes over time. Type II diabetics also experience physiological changes across time as a result of their diabetes. However, the physiological changes seem more gradual and less dramatic than that in Type I diabetics. As a consequence, Type II diabetics appear to undergo much less psychological disruption than Type I diabetics across time.

Limitations and Delimitations

1. Our diabetic subjects may have differed in several respects from that of diabetics examined in other diabetic

studies. First of all, our study was limited to diabetic subjects attending one diabetic clinic in one city. Therefore, we don't know in what significant ways these diabetics may have differed from other diabetics not attending a clinic program, or from individual's attending other clinics in other cities or countries. Dunn & Turtle (1981), report that patients who attend specialized diabetic clinics have been shown to be different from the diabetic population, with a bias toward the lower socioeconomic groups, and psychological dependency. Secondly, most of our diabetic subjects were attending an outpatient diabetic program on the referral of their physician. This also introduces the possibility of bias in terms of which subjects are included in our study. Third, our diabetic subjects had an average glycosylated hemoglobin level of 7.5 +/-2.1 millimoles/Litre. This is much lower than that normally reported in most studies. The homogeneity among glycosylated hemoglobin levels makes it more difficult for us to find statistically significant correlations when looking at metabolic control. Fourth, the very small size of our recently diagnosed Type I group does not allow us to make proper statistical analysis or statistical comparisons with the other diabetic groups. The unexpected characteristics of our recently diagnosed Type I group, makes the representativeness of this

group highly questionable. Therefore, any generalizations made on the basis of our recently diagnosed Type I group, must be cautiously guarded.

2. This study did not utilize a control group. Comparisons were made between diabetic types and groups and therefore all findings were relative in terms of diabetes. No comparisons were made between diabetics and non-diabetics.

3. Self-report measures were utilized in this study. As a result, conclusions made on the basis of this study's findings depended largely upon what our subjects were willing to reveal about themselves and/or in terms of their self-perceptions. However, self-report measures are the only means which allow us to examine diabetes from the perspective of the diabetic patient.

4. No corrections were made for the very large number of correlations which were calculated in this study. This greatly increases the possibility of making a type one error, or of finding significance when there is none.

5. In some cases, patient data was missing or unavailable with respect to some of the physiological variables. Correlations and statistical tests were conducted on the basis of data that was available. Therefore, we do not know how our results may have differed if this information had been available and utilized in our correlational and

statistical analysis.

6. No adjustment was made with respect to the age of the subjects. This confounds the interpretation of our results as no distinction was made between changes across time which were due to the normal aging process from changes which were due to diabetic duration. In addition, several research studies have indicated the importance of age of diabetic onset in terms of adaptation, prognosis and development of secondary complications.

7. This study involved four diabetic groups with each diabetic group consisting of different individuals. As a result, the comparisons which were made between recently diagnosed and long duration diabetics were made on the basis of general group differences across time rather than individual changes that occurred over time.

Implications and Recommendations

1. Because the development of severe secondary complications appears to produce great psychological discomfort for diabetic individuals it is recommended that attempts be made to psychologically prepare diabetics in advance for the likely possibility that such physical complications will eventually develop. Diabetic individuals should be well-informed in advance that secondary complications are likely to arise as a result of their illness.

2. Physicians need to be aware of the serious negative impact that retinopathy has on the individual's lifestyle. Such awareness may assist the physician with the care and treatment of the diabetics with retinopathy for diabetes and illnesses other than diabetes.

3. As there appears to be a significant relationship in Type I diabetics between maintaining metabolic control and psychological wellbeing, Type I diabetics should be strongly encouraged to adhere to their diabetic regimens and to avoid serious episodes of hyperglycemia.

4. As Type II diabetics appear to feel worse psychologically when glycosylated hemoglobin levels are low, Type II diabetics should be strongly encouraged to follow their diabetic regimens and maintain adequate metabolic control.

Suggestions for Further Research

1. A longitudinal study examining the pattern of individual changes that occur across time in diabetics would be useful.

2. Further studies examining the relationship between metabolic control and psychological wellbeing is needed in order to determine more fully diabetic Type differences and the influence of glycosylated hemoglobin levels on psychological wellbeing.

3. Studies examining the impact of secondary complications

from the diabetics' psychological and social perspective.

4. Further studies exploring the relationship between psychiatric disorder and the physical complications of diabetes would be useful.

5. Further studies of both Type I and Type II diabetics which examine the relationship between metabolic control and development of secondary complications are needed.

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APPENDIX A.

Following are the criteria that were used by the nurses at the Edmonton General Hospital clinic for the coding of obesity, vascularity, retinopathy, neuropathy, and nephropathy:

Obesity:

0 = Ideal Weight Range
1 = 10 to 20% Above Ideal Weight
2 = > 20% Above Ideal Weight

Vascularity:

0 = Post Tibial, Dorsal Pedal Pulses Normal, Feet Warm, Pink, Toenails Normal Growth
1 = Moderately Impaired (Weak) Pedal Pulses (Any of Four), Toenails Thickened, Slow Growing, Slow Healing Trauma, Lesions
2 = Absent Pedal Pulses (Any of Four), Dusky Rubor (Mottled) After Dangling, Ulceration, Gangrene

Retinopathy:

0 = None by Funduscopic Exam
1 = Simple Retinopathy
2 = Proliferative Retinopathy

Neuropathy:
(Peripheral)

0 = None Evident
1 = Weak Reflexes, Mild Sensations
2 = Absence of Reflexes and Sensations

Nephropathy:

0 = Absence of Proteinuria
1 = Proteinuria (trace to 300 mgm.)
2 = Proteinuria > 300 mgm and/or creatinine > 1.4 mmol/litre

Note: From Diabetes, Yearly Assessment (p. II-B-12) Edmonton: General Hospital (Grey Nuns) of Edmonton.

APPENDIX B

CORRELATIONAL TABLES FOR THE TOTAL FOUR GROUPS

	AGE	SEX	CONTROL	OBESITY	VASCULAR	NUPATHY
AGE	1.00	.07	.07	.29**	.42**	.28**
SEX	.07	1.00	.01	.06	-.03	-.18*
CONTROL	.07	.01	1.00	.03	.06	.24**
OBESITY	.29**	.06	.03	1.00	.17*	.24**
VASCULAR	.42**	-.03	.06	.17*	1.00	.28**
NUPATHY	.28**	-.18	.24**	.24**	.28**	1.00
NPPATHY	.05	-.17	.20*	.11	.22**	.38**
RTPATHY	-.13	-.06	.19*	-.13	-.05	.30**
HbA1c	-.16	-.10	.33**	-.04	-.04	.14
Ca9	-.01	-.01	-.10	-.06	-.08	-.18
AFFECT	.00	-.01	-.05	-.11	-.03	-.19*
WELLBEING	.00	-.01	-.09	-.10	-.05	-.19*
IHLC	-.17*	-.18*	-.12	.06	-.09	-.15
POLC	.21**	-.05	-.15*	-.03	-.14	-.10
CHLC	.15*	.05	.01	-.05	.04	-.00
SES	-.07	-.13	-.02	-.03	-.05	-.12

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR THE TOTAL FOUR GROUPS

	AGE	SEX	CONTROL	OBESITY	VASCULAR	NUPATHY
HYP	.21**	.05	.22**	.20*	.12	.22**
DEP	-.09	.01	.08	.08	-.05	.10
DEN	.33**	-.12	.01	.00	.23**	.02
INP	-.20*	-.15*	-.03	.01	-.07	.01
ALN	-.14	-.10	-.02	.02	-.16*	-.02
PID	.01	.13	-.13	.12	-.01	-.05
ANX	-.03	.25**	-.02	-.02	.06	-.02
THD	.15*	.24**	.02	.13	.02	-.12
IME	-.24**	-.06	.02	.14	-.13	.07
SOI	-.12	-.00	-.04	.01	-.04	.05
SDP	-.02	.07	.10	.02	.00	.12
DEV	-.07	.13	-.04	.05	-.08	.03
AMBUL	.38**	.01	.08	.13	.12	.14
ALERT	.03	.06	.10	.08	-.04	.07
COMMUN	.02	-.05	.07	-.11	-.14	-.04
WORK	-.10	-.09	.20*	.03	-.13	.06
RECREAT	.11	-.08	.09	.11	-.05	.05
EATING	.01	-.03	-.15	-.15	-.07	-.18
SLEEP	.18*	-.04	.08	.20*	.02	.18*
EMOTION	-.10	.06	.16*	.05	-.09	.07
BODYCARE	.28**	.01	.00	.14	.08	.12

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR THE TOTAL FOUR GROUPS

	AGE	SEX	CONTROL	OBESITY	VASCULAR	NUPATHY
HOME	.25**	.05	.09	.24**	-.00	.12
MOBILITY	.21**	-.04	.14	.02	.06	.29**
SOCIAL	-.10	-.02	.01	.03	-.12	.04
PHYSSIP	.34**	-.00	.07	.13	.10	.19*
PSYCHSIP	-.03	.02	.07	.03	-.09	.07
TOTSIP	.14	-.01	.09	.10	-.04	.13

	HbA1c	Ca9	AFFECT	WELLBEING
HbA1c	1.00	.07	-.03	.02
Ca9	.07	1.00	.77**	.94**
AFFECT	-.03	.77**	1.00	.94**
WELLBEING	.02	.94**	.94**	1.00
IHLC	-.02	.16*	.10	.14
POLC	.03	.02	-.02	.00
CHLC	.08	-.07	-.17	-.13
SES	-.05	.52**	.65**	.62**
NPPATHY	.28**	-.12	-.11	-.11
RTPATHY	.05	-.15*	-.25**	-.21*

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR THE TOTAL FOUR GROUPS

	HbA1c	Ca9	AFFECT	WELLBEING
HYP	.00	-.37**	-.39**	-.40**
DEP	.09	-.60**	-.75**	-.72**
DEN	-.02	.13	.15*	.15*
INP	.09	-.32**	-.40**	-.38**
ALN	.10	-.17*	-.21**	-.20*
PID	.01	-.32**	-.43**	-.40**
ANX	.01	-.44**	-.49**	-.40**
THD	-.08	-.28**	-.41**	-.36**
IME	.13	-.25**	-.33**	-.31**
SOI	.05	-.28**	-.44**	-.38**
SDP	.01	-.45**	-.54**	-.53**
DEV	.02	-.29**	-.46**	-.40**
AMBUL	.08	-.11	-.15*	-.13
ALERT	.07	-.28**	-.43**	-.38**
COMMUN	.10	-.24**	-.37**	-.33**
WORK	.08	-.24**	-.26**	-.26**
RECREAT	.08	-.09	-.23**	-.16*
EATING	-.05	-.13	-.14	-.14
SLEEP	.03	-.25**	-.42**	-.36**
EMOTION	.05	-.36**	-.51**	-.46**
BODYCARE	-.04	-.16*	-.16*	-.17*

* $p < .05$
** $P < .01$

CORRELATIONAL TABLES FOR THE TOTAL FOUR GROUPS

	HbA1c	Ca9	AFFECT	WELLBEING
HOME	.04	-.32	-.37**	-.37**
MOBILITY	.06	-.20	-.33**	-.28**
SOCIAL	.03	-.34**	-.54**	-.47**
PHYSSIP	.03	-.17*	-.22**	-.57**
PSYCHSIP	.05	-.37**	-.57**	-.50**
TOTSIP	.04	-.35**	-.52**	-.46**

	NPPATHY	RTPATHY	IHLC	POLC	CHLC	SES
NPPATHY	1.00	.25**	-.04	-.08	-.05	-.12
RTPATHY	.25**	1.00	-.04	.00	-.06	-.03
IHLC	-.04	-.04	1.00	-.05	-.23**	.09
POLC	-.08	.00	-.05	1.00	.30**	-.04
CHLC	-.05	-.06	-.23**	.30**	1.00	-.23**
SES	-.12	-.03	.09	-.04	-.23**	1.00
HYP	.20*	.17*	-.08	.09	.17*	-.32**
DEP	.11	.15*	-.01	-.00	.06	-.60**
DEN	-.01	-.13	-.08	.22*	-.03	.23**
INP	.10	.17*	-.09	.12	.20*	-.38**
ALN	.02	.01	-.03	-.02	.12	-.20*
PID	.11	.09	-.04	.10	.24**	-.44**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR THE TOTAL FOUR GROUPS

	NPPATHY	RTPATHY	IHLC	POLC	CHLC	SES
ANX	.05	.10	-.09	.12	.15*	-.52**
THD	-.09	-.01	-.06	.07	.25**	-.43**
IME	.16*	.10	.05	.02	.12	-.42**
SOI	.03	.07	-.02	-.08	.02	-.43**
SDP	.10	.17*	-.07	-.01	-.01	-.67**
DEV	.08	.21*	.02	.09	.07	-.44**
AMBUL	.07	.10	-.16	.27**	.08	-.15*
ALERT	.00	.15**	-.12	.10	.20**	-.37**
COMMUN	-.17*	.25**	-.08	.23**	.10	-.24**
WORK	.04	.18*	-.06	.10	.00	-.20*
RECREAT	-.06	.20*	-.10	.18*	.04	-.23**
EATING	-.13	-.09	-.09	.04	.16*	-.14*
SLEEP	.08	-.04	.04	.03	.14*	-.53**
EMOTION	.06	.19*	-.09	.06	.15	-.46**
BODYCARE	-.01	.02	-.05	.19*	.09	-.17
HOME	-.05	.06	-.14	.14*	.19*	-.30**
MOBILITY	.05	.26**	-.09	.14	.04	-.24**
SOCIAL	.03	.27**	-.04	.11	.09	-.44**
PHYSSIP	.04	.12	-.10	.22**	.08	-.20**
PSYCHSIP	-.01	.25	-.12	.17*	.18*	-.46**
TOTSIP	.00	.21*	-.13	.22**	.17*	-.45**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR THE TOTAL FOUR GROUPS

	AMBUL	ALERT	COMMUN	WORK	RECREAT
AMBUL	1.00	.25**	.29**	.27**	.48**
ALERT	.25**	1.00	.54**	.51**	.50**
COMMUN	.29**	.54**	1.00	.43**	.54**
WORK	.27**	.51**	.43**	1.00	.60**
RECREAT	.48**	.50**	.54**	.60**	1.00
EATING	.02	.13	.29**	.02	.21**
SLEEP	.37**	.43**	.35**	.31**	.33**
EMOTION	.22**	.65**	.43**	.54**	.50**
BODYCARE	.72**	.27**	.30**	.36**	.41**
HOME	.57**	.43**	.42**	.29**	.52**
MOBILITY	.55**	.28**	.44**	.34**	.43**
SOCIAL	.25**	.68**	.59**	.53**	.60**
PHYSSIP	.87**	.30**	.37**	.37**	.50**
PSYCHSIP	.30**	.87**	.69**	.60**	.63**
TOTSIP	.59**	.78**	.68**	.68**	.75**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR THE TOTAL FOUR GROUPS

	EATING	SLEEP	EMOTION	BODYCARE	HOME
AMBUL	.02	.37**	.22**	.72**	.57**
ALERT	.13	.43**	.65**	.27**	.43**
COMMUN	.29**	.35**	.43**	.30**	.42**
WORK	.02	.31**	.54**	.36**	.29**
RECREAT	.21**	.33**	.50**	.41**	.52**
EATING	1.00	-.01	.12	-.01	.07
SLEEP	-.01	1.00	.51**	.47**	.43**
EMOTION	.12	.51**	1.00	.32**	.44**
BODYCARE	-.01	.47**	.32**	1.00	.57**
HOME	.07	.43**	.44**	.57**	1.00
MOBILITY	.01	.48**	.36**	.68**	.61**
SOCIAL	.19	.45**	.73**	.30**	.43*
PHYSSIP	.00	.49**	.34**	.94**	.65**
PSYCHSIP	.22**	.50**	.83**	.35**	.50**
TOTSIP	.19*	.63**	.79**	.65**	.68**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR THE TOTAL FOUR GROUPS

	MOBILITY	SOCIAL	PHYSSIP	PSYCHSIP	TOTSIP
AMBUL	.55**	.25**	.87**	.30**	.59**
ALERT	.28**	.68**	.30**	.87**	.78**
COMMUN	.44**	.59**	.37**	.69**	.68**
WORK	.34**	.53**	.37**	.60**	.68**
RECREAT	.43**	.60**	.50**	.63**	.75**
EATING	.01	.19	.00	.22**	.19*
SLEEP	.48**	.45**	.49**	.50**	.63**
EMOTION	.36**	.73**	.34**	.83**	.79**
BODYCARE	.68**	.30**	.94**	.35**	.65**
HOME	.61**	.43*	.65**	.50**	.68**
MOBILITY	1.00	.34**	.80**	.41**	.65**
SOCIAL	.34**	1.00	.33**	.93**	.84**
PHYSSIP	.80**	.33**	1.00	.39**	.71**
PSYCHSIP	.41**	.93**	.39**	1.00	.91**
TOTSIP	.65**	.84**	.71**	.91**	1.00

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR THE TOTAL FOUR GROUPS

	AMBUL	ALERT	COMMUN	WORK	RECREAT
HYP	.48**	.44**	.39**	.38**	.42**
DEP	.18*	.57**	.45**	.40**	.36**
DEN	.12	-.19*	-.06	-.24**	-.11
IPS	.15*	.35**	.25**	.32**	.28**
ALN	.05	.21**	.18*	.12	.08
PID	.03	.46**	.30**	.18	.20*
ANX	.06	.40**	.22*	.20**	.21**
THD	.04	.44**	.24**	.18*	.14*
IME	.01	.40**	.14*	.27**	.17*
SOI	-.01	.26**	.20**	.04	.13
SDP	.02	.34**	.19*	.22**	.15*
DEV	.13	.51**	.35**	.30**	.31**

	EATING	SLEEP	EMOTION	BODYCARE	HOME
HYP	.05	.52**	.49**	.46**	.43**
DEP	.17*	.55*	.67**	.21**	.37**
DEN	-.02	-.05	-.21**	.13	.04
INP	.28**	.31**	.37**	.18*	.21**
ALN	.23**	.32**	.17*	.28**	.17*
PID	.22**	.34**	.47**	.19*	.29**
ANX	.08	.29**	.45**	.07	.12

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR THE TOTAL FOUR GROUPS

	EATING	SLEEP	EMOTION	BODYCARE	HOME
THD	.11	.34**	.40**	.25**	.24**
IME	.13	.30**	.36**	.14	.09
SOI	.17*	.22**	.28**	.05	.12
SDP	.11	.30**	.47**	.07	.10
DEV	.12	.46**	.53**	.35**	.19*

	MOBILITY	SOCIAL	PHYSSIP	PSYCHSIP	TOTSIP
HYP	.38**	.44**	.50**	.49**	.60**
DEP	.30**	.61**	.25**	.68**	.63**
DEN	.12	-.18*	.14*	-.19*	-.10
INP	.27**	.48**	.22**	.45**	.44**
ALN	.28**	.22**	.23**	.22**	.26**
PID	.14*	.51**	.14*	.53**	.45**
ANX	.05	.42**	.07	.44**	.36**
THD	.13	.41**	.17*	.44**	.39**
IME	.10	.37**	.10	.38**	.33**
SOI	.15*	.28**	.06	.31**	.25**
SDP	.19*	.37**	.09	.44**	.36**
DEV	.33**	.54**	.31**	.58**	.56**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR THE TOTAL FOUR GROUPS

	HYP	DEP	DEN	INP	ALN	PID
HYP	1.00	.52**	.02	.31**	.19*	.36**
DEP	.52**	1.00	-.19	.48**	.42**	.57**
DEN	.02	-.19	1.00	-.27**	-.12	-.18*
INP	.31**	.48**	-.27**	1.00	.51**	.45**
ALN	.19*	.42**	-.12	.51**	1.00	.50**
PID	.36**	.57**	-.18*	.45**	.50**	1.00
ANX	.42**	.53**	-.31**	.37**	.11	.49**
THD	.40**	.52**	-.12	.34**	.38**	.61**
IME	.25**	.46**	-.47**	.64**	.51**	.42**
SOI	.25**	.56**	.11	.32**	.27**	.41**
SDP	.23**	.61**	-.09	.32**	.19*	.43**
DEV	.51**	.63**	-.10	.40**	.52**	.60**

* P < .05
** P < .01

CORRELATIONAL TABLES
FOR THE TOTAL FOUR GROUPS

	ANX	THD	IME	SOI	SDP	DEV
HYP	.42**	.40**	.25**	.25**	.23**	.51**
DEP	.53**	.52**	.46**	.56**	.61**	.63**
DEN	-.31**	-.12	-.47**	.11	-.09	-.10
INP	.37**	.34**	.64**	.32**	.32**	.40**
ALN	.11	.38**	.51**	.27**	.19*	.52**
PID	.49**	.61**	.42**	.41**	.43**	.60**
ANX	1.00	.53**	.38**	.31**	.49**	.54**
THD	.53**	1.00	.45**	.38**	.47**	.65**
IME	.38**	.45**	1.00	.26**	.38**	.52**
SOI	.31**	.38**	.26**	1.00	.63**	.45**
SDP	.49**	.47**	.38**	.63**	1.00	.56**
DEV	.54**	.65**	.52**	.45**	.56**	1.00

* $P < .05$
** $P < .01$

APPENDIX C
CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I, ONSET II GROUPS

		AGE	SEX	CONTROL	OBESITY	VASCULAR	NUPATHY
AGE	I,I	1.00	.69*	.64	-.07	--	--
AGE	I,II	1.00	-.15	.12	.19	.23	.42**
SEX	I,I	.69*	1.00	.41	-.76	--	--
SEX	I,II	-.15	1.00	-.02	-.29*	-.27	-.36*
CONTROL	I,I	.64	.41	1.00	.14	--	--
CONTROL	I,II	.12	-.02	1.00	.16	.02	.29*
OBESITY	I,I	-.07	-.76	.14	1.00	--	--
OBESITY	I,II	.19	-.29*	.16	1.00	.35*	.35*
VASCULAR	I,I	--	--	--	--	1.00	--
VASCULAR	I,II	.23	-.27	.02	.35*	1.00	.42**
NUPATHY	I,I	--	--	--	--	--	1.00
NUPATHY	I,II	-.42**	-.36*	.29*	.35*	.42**	1.00
NPPATHY	I,I	--	--	--	--	--	--
NPPATHY	I,II	-.00	-.36*	.30*	.15	.35*	.40**
RTPATHY	I,I	--	--	--	--	--	--
RTPATHY	I,II	.28*	-.23	.13	.10	.10	.27
HbAlc	I,I	-.29	-.57	.41	.86	--	--
HbAlc	I,II	-.09	-.30*	.30*	.02	.29*	.25
Ca9	I,I	.07	.34	-.52	-.58	--	--
Ca9	I,II	-.09	.20	.02	-.24	-.07	-.24
AFFECT	I,I	.33	.48	.13	-.26	--	--
AFFECT	I,II	-.17	.24	.09	-.22	.06	-.22
WELLBE	I,I	.22	.49	-.07	-.21	--	--
WELLBE	I,II	-.13	.24	.06	-.23	.00	-.23
IHLC	I,I	.14	-.53	-.08	.93**	--	--
IHLC	I,II	-.06	.25	-.14	-.14	-.01	-.39*
POLC	I,I	-.06	-.15	.04	.75	--	--
POLC	I,II	.11	.12	-.36*	-.06	-.23	-.08

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I,ONSET II GROUPS

		AGE	SEX	CONTROL	OBESITY	VASCULAR	NUPATHY
CHLC	I,I	.01	.46	-.56	-.72	--	--
CHLC	I,II	.10	-.17	-.02	.08	.29*	.18
SES	I,I	-.38	-.20	.33	-.15	--	
SES	I,II	.07	.14	.17	-.12	-.12	-.08
HYP	I,I	.26	.61	.41	-.69	--	--
HYP	I,II	.23	-.30*	.20	.26	.14	.41**
DEP	I,I	-.52	-.28	-.69	-.47	--	--
DEP	I,II	.03	-.22	.12	.17	.05	.20
DEN	I,I	-.31	-.47	-.02	.21	--	--
DEN	I,II	.35*	-.09	-.16	-.04	.12	.16
INP	I,I	-.31	-.52	-.68	.07	--	--
INP	I,II	-.08	-.32	.02	.07	-.03	.29*
ALN	I,I	.15	-.22	-.19	.43	--	--
ALN	I,II	-.14	-.34*	-.07	-.13	-.09	-.02
PID	I,I	.36	-.24	-.13	.43	--	--
PID	I,II	-.10	-.22	-.12	.03	.00	.00
ANX	I,I	.04	.16	.77	.15	--	--
ANX	I,II	-.09	.25	-.13	-.02	.04	.08
THD	I,I	.51	.50	.56	-.60	--	--
THD	I,II	.14	-.03	-.13	.07	.13	-.03
IME	I,I	-.27	-.38	-.23	.66	--	--
IME	I,II	-.29*	-.20	-.07	.15	.12	-.04
SOI	I,I	-.45	-.56	-.98**	-.25	--	--
SOI	I,II	-.13	-.13	-.15	.08	.17	.19
SDP	I,I	-.53	-.66	-.81*	.35	--	--
SDP	I,II	-.02	-.03	-.05	.11	.21	.11
DEV	I,I	-.01	.04	.40	.00	--	--
DEV	I,II	-.04	-.13	-.19	.08	-.02	.06

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I,ONSET II GROUPS

		AGE	SEX CONTROL	OBESITY	VASCULAR	NUPATHY
AMBUL	I,I	-.20	.36	-.25	-.54	--	--
AMBUL	I,II	.51**	-.15	.20	.24	-.10	.50**
ALERT	I,I	-.07	.31	.42	-.47	--	--
ALERT	I,II	.14	-.18	-.05	.15	-.16	-.04
COMMUN	I,I	-.18	.07	.58	-.06	--	--
COMMUN	I,II	.33*	-.06	-.08	.03	-.14	-.02
WORK	I,I	.02	.39	.42	-.53	--	--
WORK	I,II	.09	-.20	.15	.18	-.12	.10
RECREAT	I,I	-.50	.19	.06	-.70	--	--
RECREAT	I,II	.25	-.26	-.03	.21	-.15	.13
EATING	I,I	.30	.01	-.61	-.22	--	--
EATING	I,II	-.04	-.10	-.02	-.07	-.25	-.14
SLEEP	I,I	-.04	.35	.38	-.53	--	--
SLEEP	I,II	.00	-.27	.17	.14	-.19	.13
EMOTION	I,I	-.15	.38	.41	-.58	--	--
EMOTION	I,II	-.04	-.08	.19	.07	-.09	.09
BODYCARE	I,I	-.04	.35	.38	-.53	--	--
BODYCARE	I,II	.39**	-.08	-.23	-.06	-.11	.21
HOME	I,I	-.35	.17	.41	-.22	--	--
HOME	I,II	.52**	-.26	.01	.18	-.12	.33*
MOBILITY	I,I	-.04	.35	.38	-.53	--	--
MOBILITY	I,II	.51**	-.17	-.07	.02	-.14	.40**
SOCIAL	I,I	-.43	-.62	-.53	-.03	--	--
SOCIAL	I,II	.28*	-.15	-.08	-.13	.13	.16
PHYSSIP	I,I	-.12	.49	.28	-.76	--	--
PHYSSIP	I,II	.53**	-.15	-.04	.07	-.13	.41**
PSYCHSIP	I,I	-.30	-.03	.32	-.42	--	--
PSYCHSIP	I,II	.23	-.13	-.04	.10	-.14	.10

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I,ONSET II GROUPS

		AGE	SEX	CONTROL	OBESITY	VASCULAR	NUPATHY
TOTSIP	I,I	-.25	.16	.32	-.54	--	--
TOTSIP	I,II	.36*	-.19	-.03	.14	-.16	.21

		HbAlc	Ca9	AFFECT	WELLBEING
HbAlc	I,I	1.00	-.93*	-.75	-.96*
HbAlc	I,II	1.00	-.11	-.02	-.06
Ca9	I,I	-.93*	1.00	.28	.81*
Ca9	I,II	-.11	1.00	.91**	.98**
AFFECT	I,I	-.75	.28	1.00	.79*
AFFECT	I,II	-.02	.91**	1.00	.98**
WELLBEING	I,I	-.96*	.81*	.79*	1.00
WELLBEING	I,II	-.06	.98**	.98**	1.00
IHLC	I,I	.42	-.03	-.47	-.29
IHLC	I,II	-.21	.29*	.37*	.34*
POLC	I,I	.61	.66	.54	.92**
POLC	I,II	-.19	-.24	-.20	-.23
CHLC	I,I	-.86*	.37	.76*	.73*
CHLC	I,II	.22	-.10	-.20	-.15
SES	I,I	.23	.18	-.32	-.05
SES	I,II	-.06	.66**	.66**	.67**
NPPATHY	I,I	--	--	--	--
NPPATHY	I,II	.42**	-.05	.02	-.01
RTPATHY	I,I	--	--	--	--
RTPATHY	I,II	-.09	-.25	-.29*	-.27
HYP	I,I	-.51	.10	.52	.36
HYP	I,II	.39**	-.60**	-.48**	-.55**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I,ONSET II GROUPS

		HbA1c	Ca9	AFFECT	WELLBEING
DEP	I,I	-.29	-.32	-.72*	-.72
DEP	I,II	.32*	-.84**	-.80*	-.84**
DEN	I,I	.14	.23	-.58	-.20
DEN	I,II	.17	.08	-.03	.02
INP	I,I	-.38	-.60	-.08	-.49
INP	I,II	.16	-.51**	-.48**	-.52**
ALN	I,I	-.02	-.56	.30	-.18
ALN	I,II	.25	-.02	-.05	-.04
PID	I,I	-.56	-.04	-.15	-.16
PID	I,II	.28*	-.61**	-.56**	-.60**
ANX	I,I	.53	.27	.27	.48
ANX	I,II	.07	-.67**	-.55**	-.62**
THD	I,I	-.46	-.54	.03	-.41
THD	I,II	.10	-.48**	-.42**	-.45**
IME	I,I	.29	-.51	.38	-.06
IME	I,II	.16	-.41**	-.33*	-.38*
SOI	I,I	-.29	-.50	-.74*	-.83*
SOI	I,II	.09	-.53**	-.59**	-.57**
SDP	I,I	-.14	.17	.05	.14
SDP	I,II	-.03	-.61**	-.58**	-.60**
DEV	I,I	-.09	-.07	.70*	.41
DEV	I,II	.11	-.45**	-.44**	-.45**
AMBUL	I,I	N/A	.11	-.29	-.13
AMBUL	I,II	.00	-.12	-.18	-.15
ALERT	I,I	-.20	.11	.52	.39
ALERT	I,I	.08	-.58**	-.64**	-.62**
COMMUN	I,I	.29	.11	.39	.39
COMMUN	I,II	.03	-.53**	-.54**	-.55**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I,ONSET II GROUPS

		HbA1c	Ca9	AFFECT	WELLBEING
WORK	I,I	-.30	.06	.57	.38
WORK	I,II	.12	-.54**	-.47**	-.51**
RECREAT	I,I	.00	.07	.08	.10
RECREAT	I,II	.01	-.60**	-.65**	-.63**
EATING	I,I	-.57	.05	-.46	-.39
EATING	I,I	.11	-.41**	-.43**	-.43**
SLEEP	I,I	-.29	.11	.54	.39
SLEEP	I,II	.28*	-.36*	-.34*	-.35*
EMOTION	I,I	-.10	.14	.43	.35
EMOTION	I,II	.17	-.67**	-.60**	-.65**
BODYCARE	I,I	-.29	.11	.54	.39
BODYCARE	I,II	-.02	-.13	-.18	-.15
HOME	I,I	.56	.17	.04	.21
HOME	I,I	-.00	-.39**	-.48**	-.45**
MOBILITY	I,I	-.29	.11	.54	.39
MOBILITY	I,II	-.03	-.24	-.35	-.30*
SOCIAL	I,I	-.23	-.50	-.57	-.71
SOCIAL	I,II	-.03	-.73**	-.72**	-.74**
PHYSSIP	I,I	-.29	.15	.42	.33
PHYSSIP	I,II	-.02	-.18	-.26	-.22
PSYCHSIP	I,I	-.15	-.13	.17	.01
PSYCHSIP	I,II	.01	-.74**	-.74**	-.75**
TOTSIP	I,I	-.19	-.03	.28	.14
TOTSIP	I,II	-.01	-.67*	-.69**	-.69**

		NPPATHY	RTPATHY	IHLC	POLC	CHLC	SES
NPPATHY	I,I	1.00	--	--	--	--	
NPPATHY	I,II	1.00	.40**	-.34**	-.19	.16	.02

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I, ONSET II GROUPS

		NPPATHY	RTPATHY	IHLC	POLC	CHLC	SES
RTPATHY	I,I	--	1.00	--	--	--	--
RTPATHY	I,II	.40**	1.00	-.02	.32*	.14	-.03
IHLC	I,I	--	--	1.00	.20	-.65	-.19
IHLC	I,II	-.34**	-.02	1.00	.02	-.35*	.15
POLC	I,I	--	--	.20	1.00	.15	.00
POLC	I,II	-.19	.32*	.03	1.00	.21	-.02
CHLC	I,I	--	--	-.65	.15	1.00	-.39
CHLC	I,II	.16	.14	-.35*	.21	1.00	-.04
SES	I,I	--	--	-.19	.00	-.39	1.00
SES	I,II	.02	-.03	.15	-.02	-.04	1.00
HYP	I,I	--	--	-.77*	-.12	.42	.39
HYP	I,II	.26	.32*	-.10	.14	.22	-.34*
DEP	I,I	--	--	-.04	-.75*	-.10	.08
DEP	I,II	.04	.21	-.24	.06	.1	-.65**
DEN	I,I	--	--	.37	-.05	-.58	.78
DEN	I,II	-.12	-.22	-.10	.12	.26	.04
INP	I,I	--	--	.15	-.32	.18	-.33
INP	I,II	.04	.29*	-.16	.27	.17	-.47**
ALN	I,I	--	--	.26	.05	.20	-.79
ALN	I,II	.02	-.16	.00	-.07	.09	-.19
PID	I,I	--	--	.64	-.10	-.26	-.08
PID	I,II	.17	.15	-.12	.16	.05	-.55**
ANX	I,I	--	--	-.28	.51	-.20	.60
ANX	I,II	.06	.32*	-.07	.31	-.03	-.57**
THD	I,I	--	--	-.36	-.68*	-.01	.11
THD	I,II	-.01	.20	.12	-.01	-.06	-.44**
IME	I,I	--	--	-.01	.31	.26	-.49
IME	I,II	.18	.17	-.08	.07	.23	-.55**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I,ONSET II GROUPS

		NPPATHY	RTPATHY	IHLC	POLC	CHLC	SES
SOI	I,I	--	--	.23	-.70*	-.35	.22
SOI	I,II	-.02	-.06	-.27	-.28*	-.07	-.62**
SDP	I,I	--	--	.35	.37	.24	-.21
SDP	I,II	-.03	.06	-.04	-.12	-.24	-.76**
DEV	I,I	--	--	-.44	.45	.31	.27
DEV	I,II	.13	.18	.10	.07	-.13	-.53**
AMBUL	I,I	--	--	-.40	-.42	.18	-.10
AMBUL	I,II	.04	.18	-.38	.18	.18	.11
ALERT	I,I	--	--	-.76*	.17	.34	.57
ALERT	I,II	-.10	.28	-.12	.14	.10	-.41**
COMMUN	I,I	--	--	-.52	.43	.04	.65
COMMUN	I,II	-.20	.25	-.01	.43**	.06	-.37*
WORK	I,I	--	--	-.79*	.10	.40	.47
WORK	I,II	.04	.26	-.09	.20	.08	-.38**
RECREAT	I,I	--	--	-.85**	-.12	.29	.55
RECREAT	I,II	-.04	.31*	-.25	.39**	.17	-.51**
EATING	I,I	--	--	.49	-.53	-.17	-.24
EATING	I,II	-.27	.22	-.18	-.28*	.15	-.38**
SLEEP	I,I	--	--	-.79*	.11	.39	.53
SLEEP	I,II	-.10	-.01	-.02	.05	.04	-.40*
EMOTION	I,I	--	--	-.85**	.11	.39	.58
EMOTION	I,II	.11	.22	-.21	.20	.03	-.53**
BODYCARE	I,I	--	--	-.79*	.11	.34	.53
BODYCARE	I,II	-.15	-.05	-.05	.20	.09	-.07
HOME	I,I	--	--	-.58	.11	.39	.50
HOME	I,II	-.08	.16	-.21	.32*	.25	-.25
MOBILITY	I,I	--	--	-.79	.20	.39	.53
MOBILITY	I,II	-.12	.06	-.14	.18	-.03	-.35*

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I, ONSET II GROUPS

		NPPATHY	RTPATHY	IHLC	POLC	CHLC	SES
SOCIAL	I,I	--	--	.19	-.52	-.38	.42
SOCIAL	I,II	.01	.32*	-.24	.47**	.14	-.54**
PHYSSIP	I,I	--	--	-.93**	-.05	.45	.49
PHYSSIP	I,II	-.09	.07	-.21	.21	.10	-.10
PSYCHSIP	I,I	--	--	-.60	-.07	.09	.74*
PSYCHSIP	I,II	-.02	.31*	-.24	.41**	.11	-.55**
TOTSIP	I,I	--	--	-.75*	-.05	.23	.69*
TOTSIP	I,II	-.04	.28*	-.26	.42**	.13	-.50**

		AMBUL	ALERT	COMMUN	WORK	RECREAT
AMBUL	I,I	1.00	-.18	-.25	-.18	.50
AMBUL	I,II	1.00	.37*	.24*	.19	.32*
ALERT	I,I	-.18	1.00	.89**	.99**	.69*
ALERT	I,II	.37*	1.00	.64**	.75**	.78**
COMMUN	I,I	-.25	.89**	1.00	.83*	.63
COMMUN	I,II	.24	.64**	1.00	.61**	.76**
WORK	I,I	-.18	.99**	.83*	1.00	.65
WORK	I,II	.19	.75**	.61**	1.00	.81**
RECREAT	I,I	.50	.69*	.63	.65	1.00
RECREAT	I,II	.32*	.78**	.76**	.81**	1.00
EATING	I,I	.02	-.62	-.83	-.56	-.58
EATING	I,II	.08	.39**	.59**	.41**	.71**
SLEEP	I,I	-.17	1.00**	.84**	1.00**	.69*
SLEEP	I,II	.37*	.50**	.42**	.41**	.54**
EMOTION	I,I	.09	.96**	.87**	.94**	.85**
EMOTION	I,II	.28*	.64**	.53**	.58**	.73**
BODYCARE	I,I	-.17	1.00**	.84**	1.00**	.69*
BODYCARE	I,II	.62*	.44**	.33**	.26	.33*

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I, ONSET II GROUPS

		AMBUL	ALERT	COMMUN	WORK	RECREAT
HOME	I,I	.47	.53	.71*	.46	.83**
HOME	I,II	.70**	.58**	.63**	.39**	.71**
MOBILITY	I,I	-.17	1.00**	.84**	1.00**	.69*
MOBILITY	I,II	.61**	.45**	.54**	.32*	.58**
SOCIAL	I,I	-.29	-.02	-.10	-.03	-.01
SOCIAL	I,II	.43**	.85**	.74**	.79**	.89**
PHYSSIP	I,I	.23	.91**	.73*	.91**	.87**
PHYSSIP	I,II	.85**	.48**	.41**	.29*	.45**
PSYCHSIP	I,I	-.26	.89**	.78*	.87**	.66
PSYCHSIP	I,II	.42**	.91**	.79**	.79**	.89**
TOTSIP	I,I	-.11	.95**	.82	.94**	.77*
TOTSIP	I,II	.55**	.90**	.79**	.80**	.91**

		EATING	SLEEP	EMOTION	BODYCARE	HOME
AMBUL	I,I	.02	-.17	.09	-.17	.47
AMBUL	I,II	.08	.37*	.28*	.62**	.70**
ALERT	I,I	-.62	1.00**	.96**	1.00**	.53
ALERT	I,II	.39**	.50**	.64**	.44**	.58**
COMMUN	I,I	-.83*	.84**	.87**	.84**	.71*
COMMUN	I,II	.59**	.42**	.53**	.33*	.63**
WORK	I,I	-.56	1.00**	.94**	1.00**	.46
WORK	I,II	.41**	.41**	.58**	.26	.39**
RECREAT	I,I	-.58	.69*	.85**	.69*	.83**
RECREAT	I,II	.71**	.54**	.73**	.33*	.71**
EATING	I,I	1.00	-.56	-.68*	-.56	-.77
EATING	I,II	1.00	.48**	.60**	.18	.50**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I, ONSET II GROUPS

		EATING	SLEEP	EMOTION	BODYCARE	HOME
SLEEP	I,I	-.56	1.00	.95**	1.00*	.48
SLEEP	I,II	.48**	1.00	.59**	.31*	.62**
EMOTION	I,I	-.68	.95**	1.00	.95**	.73*
EMOTION	I,II	.60**	.59**	1.00	.17	.46**
BODYCARE	I,I	-.56	1.00*	.95**	1.00	.48
BODYCARE	I,II	.18	.31*	.17	1.00	.58**
HOME	I,I	-.77	.48	.73*	.48	1.00
HOME	I,II	.50**	.62**	.46**	.58**	1.00
MOBILITY	I,I	-.56	1.00*	.95**	1.00*	.48
MOBILITY	I,II	.40**	.47**	.32*	.74**	.80**
SOCIAL	I,I	.37	-.00	-.13	-.00	-.34
SOCIAL	I,II	.56**	.60	.76**	.51**	.71**
PHYSSIP	I,I	-.54	.92**	.97**	.92**	.66
PHYSSIP	I,II	.23	.43**	.28*	.91**	.78**
PSYCHSIP	I,I	-.43	.89**	.82*	.89**	.39
PSYCHSIP	I,II	.59**	.59**	.83**	.45**	.69**
TOTSIP	I,I	-.51	.95**	.92**	.95**	.51
TOTSIP	I,II	.63**	.66**	.81**	.58**	.80**

		MOBILITY	SOCIAL	PHYSSIP	PSYCHSIP	TOTSIP
AMBUL	I,I	-.17	-.29	.23	-.26	-.11
AMBUL	I,II	.61**	.43**	.85**	.42**	.55**
ALERT	I,I	1.00**	-.02	.91**	.89**	.95**
ALERT	I,II	.45**	.85**	.48**	.91**	.90**
COMMUN	I,I	.84**	-.10	.73*	.78*	.82
COMMUN	I,II	.54**	.74**	.41**	.79**	.79**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I, ONSET II GROUPS

		MOBILITY	SOCIAL	PHYSSIP	PSYCHSIP	TOTSIP
WORK	I,I	1.00**	-.03	.91**	.87**	.94**
WORK	I,II	.32*	.79**	.29*	.79**	.80**
RECREAT	I,I	.69*	-.01	.87**	.66	.77*
RECREAT	I,II	.58**	.89**	.45**	.89**	.91**
EATING	I,I	-.56	.37	-.54	-.43	-.51
EATING	I,II	.40**	.56**	.23	.59**	.63**
SLEEP	I,I	1.00*	-.00	.92**	.89**	.95**
SLEEP	I,II	.47**	.56**	.43**	.59**	.66
EMOTION	I,I	.95**	-.13	.97**	.82*	.92**
EMOTION	I,II	.32*	.76**	.28*	.83**	.81**
BODYCARE	I,I	1.00*	-.00	.92**	.89**	.95**
BODYCARE	I,II	.74**	.51**	.91**	.45**	.58**
HOME	I,I	.48	-.34	.66	.39	.51
HOME	I,II	.80**	.71**	.78**	.69**	.80**
MOBILITY	I,I	1.00	-.00	.92**	.89**	.95**
MOBILITY	I,II	1.00	.62**	.87**	.58**	.73**
SOCIAL	I,I	-.00	1.00	-.12	.43	.25
SOCIAL	I,II	.62**	1.00	.58**	.97**	.97**
PHYSSIP	I,I	.92**	-.12	1.00	.77*	.90**
PHYSSIP	I,II	.87**	.58**	1.00	.54**	.69**
PSYCHSIP	I,I	.89**	.43	.77*	1.00	.97**
PSYCHSIP	I,II	.58**	.97**	.54**	1.00	.98**
TOTSIP	I,I	.95**	.25	.90**	.97**	1.00
TOTSIP	I,II	.73**	.97**	.69**	.98**	1.00

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I, ONSET II GROUPS

		AMBUL	ALERT	COMMUN	WORK	RECREAT
HYP	I,I	-.10	.89**	.62	.93**	.54
HYP	I,II	.48**	.55**	.46**	.62**	.47**
DEP	I,I	.60	-.37	-.49	-.36	.25
DEP	I,II	.20	.72**	.53**	.60**	.67**
DEN	I,I	-.28	.04	.12	-.04	-.00
DEN	I,II	.33*	.03	-.00	-.18	.00
INP	I,I	-.31	-.25	-.42	-.20	-.28
INP	I,II	.12	.45**	.26	.53**	.58**
ALN	I,I	-.35	-.37	-.37	-.29	-.55
ALN	I,II	-.03	.21	.01	.18	.19
PID	I,I	-.68*	-.31	-.42	-.28	-.76*
PID	I,II	.10	.69**	.56**	.53**	.57**
ANX	I,I	-.18	.67*	.90**	.60	.44
ANX	I,II	.03	.47**	.41**	.45**	.43**
THD	I,I	-.07	.37	.08	.46	.10
THD	I,II	.09	.57**	.34*	.41**	.38*
IME	I,I	-.26	-.04	.08	-.02	-.10
IME	I,II	-.03	.36*	.02	.34*	.27*
SOI	I,I	.05	-.31	-.41	-.31	-.03
SOI	I,II	-.04	.37*	.10	.13	.21
SDP	I,I	-.31	-.32	-.29	-.34	-.29
SDP	I,II	-.09	.40**	.26	.31*	.37*
DEV	I,I	-.60	.83*	.80*	.82*	.29
DEV	I,II	.13	.54**	.28*	.43**	.41**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I, ONSET II GROUPS

		EATING	SLEEP	EMOTION	BODYCARE	HOME
HYP	I,I	-.29	.92**	.83**	.92**	.29
HYP	I,II	.22	.66**	.58**	.51**	.58**
DEP	I,I	.44	-.33	-.23	-.33	-.05
DEP	I,II	.49**	.59**	.82**	.19	.44**
DEN	I,I	.31	.02	-.03	.02	-.06
DEN	I,II	.08	.07	-.05	.59**	.28*
INP	I,I	.33	-.21	-.39	-.21	-.65
INP	I,II	.38*	.52**	.44**	.27*	.35*
ALN	I,I	.03	-.35	-.49	-.35	-.56
ALN	I,II	.29*	.41**	.12	.43**	.10
PID	I,I	.72*	-.28	-.55	-.28	-.88**
PID	I,II	.37*	.55**	.56**	.38**	.38**
ANX	I,I	-.76	.61	.70*	.61	.74*
ANX	I,II	.21	.30*	.56**	.15	.19
THD	I,I	.22	.42	.30	.42	-.14
THD	I,II	.12	.38*	.43**	.50**	.23
IME	I,I	-.49	-.06	-.09	-.06	-.05
IME	I,II	.14	.34*	.43**	.18	.04
SOI	I,I	.55	-.28	-.33	-.28	-.36
SOI	I,II	.14	.22	.43**	.16	.06
SDP	I,I	.16	-.31	-.41	-.31	-.44
SDP	I,II	.26	.17	.55**	.09	.10
DEV	I,I	-.67	.80*	.67*	.80*	.22
DEV	I,II	.18	.40**	.51**	.50**	.20

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I, ONSET II GROUPS

		MOBILITY	SOCIAL	PHYSSIP	PSYCHSIP	TOTSIP
HYP	I,I	.92**	-.03	.87**	.76*	.84**
HYP	I,II	.48**	.66**	.56**	.64**	.67**
DEP	I,I	-.33	.56	-.09	-.06	-.09
DEP	I,II	.32*	.72**	.26	.79**	.74*
DEN	I,I	.02	.63	-.09	.34	.20
DEN	I,II	.42**	.09	.51**	.06	.16
INP	I,I	-.21	.65	-.33	.03	-.11
INP	I,II	.35*	.59**	.28*	.51**	.52**
ALN	I,I	-.35	-.01	-.49	-.36	-.43
ALN	I,II	.27*	.18	.26	.13	.16
PID	I,I	-.28	.48	-.55	-.12	-.29
PID	I,II	.37*	.68**	.32	.70**	.66**
ANX	I,I	.61	-.33	.53	.50	.55
ANX	I,II	.24	.58**	.15	.58**	.51**
THD	I,I	.42	.29	.39	.43	.43
THD	I,II	.32*	.56**	.35*	.54**	.50**
IME	I,I	-.06	-.07	-.16	-.06	-.10
IME	I,II	.02	.36*	.07	.34*	.27
SOI	I,I	.28	.92**	-.26	.14	-.01
SOI	I,II	.22	.29*	.12	.35*	.29*
SDP	I,I	-.31	.26	-.43	-.18	-.28
SDP	I,II	.29*	.44*	.10	.50**	.43**
DEV	I,I	.80*	-.02	.56	.72*	.71*
DEV	I,II	.40**	.57**	.39*	.55**	.54**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I, ONSET II GROUPS

		HYP	DEP	DEN	INP	ALN	PID
HYP	I,I	1.00	-.28	-.05	-.22	-.34	.13
HYP	I,II	1.00	.69**	.26	.53**	.28*	.65**
DEP	I,I	-.28	1.00	-.22	.40	-.16	-.09
DEP	I,II	.69**	1.00	.02	.59**	.26	.73**
DEN	I,I	-.05	.22	1.00	-.04	-.60	.45
DEN	I,II	.26	.02	1.00	-.05	.31*	.08
INP	I,I	-.22	.40	-.04	1.00	.66*	.44
INP	I,II	.53**	.59**	-.05	1.00	.58**	.61**
ALN	I,I	-.34	-.16	-.60	.66*	1.00	.24
ALN	I,II	.28*	.26	.31*	.58**	1.00	.54**
PID	I,I	-.13	-.09	.45	.44	.24	1.00
PID	I,II	.65**	.73**	.08	.61**	.54**	1.00
ANX	I,I	.43	-.61	.13	-.71*	-.46	-.41
ANX	I,II	.51**	.62**	-.08	.45**	-.03	.60**
THD	I,I	.67*	.06	-.06	.08	-.03	.21
THD	I,II	.58**	.61**	.19	.46**	.47**	.68**
IME	I,I	-.25	-.20	-.58	.51	.82*	-.20
IME	I,II	.45**	.54**	-.02	.60**	.54**	.57**
SOI	I,I	-.25	.83*	.51	.65	-.00	.37
SOI	I,II	.27*	.63**	.21	.29*	.22	.47**
SDP	I,I	-.48	.12	.16	.63	.37	.32
SDP	I,II	.25	.64**	.01	.30*	.05	.44**
DEV	I,I	.64	-.64	-.10	.04	.13	-.07
DEV	I,II	.52**	.59**	.20	.47**	.52**	.71**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE I, ONSET I AND TYPE I, ONSET II GROUPS

		ANX	THD	IME	SOI	SDP	DEV
HYP	I,I	.43	-.61	.13	-.71*	-.46	-.41
HYP	I,II	.51**	.58**	-.08	.27	.25	.52**
DEP	I,I	-.61*	.06	-.20	.82	.12	.64
DEP	I,II	.62**	.61**	.19	.63	.64**	.60**
DEN	I,I	.13	-.05	-.58	.51	.16	-.10
DEN	I,II	-.08	.20	-.02	.21	.01	.47**
INP	I,I	-.71	.08	.51	.65	.63	.04
INP	I,II	.45**	.46**	.21	.29*	.30	.47**
ALN	I,I	-.48	-.03	.82*	-.00	.37	.13
ALN	I,II	-.03	.46**	.01	.23	.05	.52**
PID	I,I	-.41	-.64	-.10	.04	.13	-.07
PID	I,II	.60**	.68**	.57**	.47**	.44	.71**
ANX	I,I	1.00	-.01	-.08	-.58	-.46	.59
ANX	I,II	1.00	.52**	.38**	.43**	.55**	.56**
THD	I,I	-.01	1.00	-.28	.20	-.66	.19
THD	I,II	.52**	1.00	.62**	.48**	.56**	.81**
IME	I,I	-.08	-.28	1.00	-.14	.41	.41
IME	I,II	.38**	.62**	1.00	.35*	.40**	.65**
SOI	I,I	-.58	.20	-.14	1.00	.26	-.39
SOI	I,II	.43**	.48**	.35*	1.00	.78**	.50**
SDP	I,I	-.46	-.66	.41	.26	1.00	.00
SDP	I,II	.55**	.56**	.40**	.78**	1.00	.61**
DEV	I,I	.59	.19	.41	-.39	.00	1.00
DEV	I,II	.56**	.81**	.65**	.50**	.61**	1.00

* $p < .05$
** $p < .01$

APPENDIX D.

CORRELATIONAL TABLES FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		AGE	SEX	CONTROL	OBESITY	VASCULAR	NUPATHY
AGE	II,I	1.00	.33*	-.00	.02	.22	.57**
AGE	II,II	1.00	.01	.11	-.38**	.36**	-.11
SEX	II,I	.33*	1.00	.15	.31	-.17	-.02
SEX	II,II	.01	1.00	-.02	.27*	.11	-.14
CONTROL	II,I	-.00	.15	1.00	-.10	-.07	.28
CONTROL	II,II	.11	-.02	1.00	.04	.13	.09
OBESITY	II,I	.02	.31	-.10	1.00	-.07	-.04
OBESITY	II,II	-.38*	.27*	.04	1.00	-.08	.24*
VASCULAR	II,I	.22	-.17	-.07	-.07	1.00	.17
VASCULAR	II,II	.36**	.11	.13	-.08	1.00	.21
NUPATHY	II,I	.57**	.02	.28	-.04	.17	1.00
NUPATHY	II,II	-.11	-.14	.09	.24*	.21	1.00
NPPATHY	II,I	-.05	.02	.20	-.08	-.18	-.05
NPPATHY	II,II	-.07	-.12	.05	.11	.22	.39**
RTPATHY	II,I	--	--	--	--	--	--
RTPATHY	II,II	.20	.09	.24	.12	.09	.43
HbA1c	II,I	.09	.16	.13	-.09	-.28	-.11
HbA1c	II,II	-.14	.04	.34**	.07	.05	.12
Ca9	II,I	-.05	-.26	-.28	-.27	-.02	-.27
Ca9	II,II	-.04	-.04	-.06	.09	-.16	-.08
AFFECT	II,I	-.04	-.24	-.20	-.22	.13	-.11
AFFECT	II,II	.05	-.12	-.09	-.08	-.16	-.17
WELLBEING	II,I	-.06	-.29*	-.26	-.27	.06	-.21
WELLBEING	II,II	.00	-.09	-.10	-.01	-.16	-.13

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		AGE	SEX	CONTROL	OBESITY	VASCULAR	NUPATHY
IHLC	II,I	-.49**	-.43**	-.11	.00	.18	-.16
IHLC	II,II	-.18	-.23*	-.12	.12	-.31*	-.07
POLC	II,I	-.02	-.09	-.28	-.20	.11	-.24
POLC	II,II	.45**	-.11	.02	-.16	.23	-.04
CHLC	II,I	.06	.03	-.07	-.02	-.28	.10
CHLC	II,II	.24*	.15	.16	-.22	.15	-.10
SES	II,I	-.13	-.32*	-.35*	-.07	.15	-.19
SES	II,II	-.07	-.20	.01	.06	-.10	-.06
HYP	II,I	-.20	.31*	.24	.26	-.25	-.02
HYP	II,II	.10	.18	.14	.02	.11	-.03
DEP	II,I	-.00	.26	.16	.32	-.18	.11
DEP	II,II	-.02	.05	.07	.07	.05	.03
DEN	II,I	.12	-.27	.03	-.23	.28	-.04
DEN	II,II	.35**	-.03	.14	-.16	.15	-.02
INP	II,I	-.19	.07	-.06	.27	-.31*	.10
INP	II,II	-.21	-.09	.06	-.04	.07	-.06
ALN	II,I	-.27	.09	-.05	.21	-.38*	-.25
ALN	II,II	-.19	.01	.09	.11	-.13	.09
PID	II,I	-.03	.25	-.28	.29	-.44*	-.10
PID	II,II	.06	.32**	-.02	.06	.20	-.08
ANX	II,I	-.04	.35*	.06	.40*	-.37*	-.10
ANX	II,II	.11	.22	.06	-.26*	.31*	-.13
THD	II,I	.04	.47**	.10	.19	-.37*	-.17
THD	II,II	.07	.29*	.18	.06	.12	-.19

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		AGE	SEX CONTROL	OBESITY	VASCULAR	NUPATHY
IME	II,I	-.10	.25	.26	.28	-.27	.12
IME	II,II	-.28	-.13	-.01	.21	-.11	.17
SOI	II,I	.09	.08	-.07	-.04	-.23	-.08
SOI	II,II	.01	.17	.19	.15	.05	.05
SDP	II,I	.26	.38*	.20	.11	-.24	.25
SDP	II,II	.11	.10	.35**	.03	.09	.10
DEV	II,I	-.06	.30*	.03	-.27	-.32*	.01
DEV	II,II	.03	.29*	.05	.11	.05	-.01
AMBUL	II,I	.18	.31	.08	.15	.07	-.07
AMBUL	II,II	.25*	.05	-.06	-.09	.03	-.12
ALERT	II,I	-.00	.17	.17	.43*	-.21	.17
ALERT	II,II	.06	.15	.17	-.06	.17	.15
COMMUN	II,I	-.02	.11	.14	.15	-.20	-.04
COMMUN	II,II	.08	-.09	.08	-.21	-.11	-.08
WORK	II,I	-.18	.15	.03	.34*	-.01	-.10
WORK	II,II	-.19	-.15	.23*	.08	-.18	.05
RECREAT	II,I	-.01	.26	.14	.47**	.01	-.05
RECREAT	II,II	.15	-.06	.04	-.02	-.09	-.06
EATING	II,I	.20	-.11	.01	-.07	-.02	.25
EATING	II,II	.03	.02	-.25*	-.29*	-.02	-.35
SLEEP	II,I	.13	.11	.18	.25	-.23	.16
SLEEP	II,II	-.04	-.02	-.02	.03	-.00	.09
EMOTION	II,I	-.10	.24	.19	.30	-.20	.05
EMOTION	II,II	-.09	.07	.10	.08	.02	.08

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		AGE	SEX	CONTROL	OBESITY	VASCULAR	NUPATHY
BODYCARE	II,I	.04	.09	.19	.21	-.04	.31*
BODYCARE	II,II	.11	.06	-.07	.08	-.01	.10
HOME	II,I	-.05	.19	-.10	.27	.37*	-.11
HOME	II,II	.09	.23*	-.00	.20	-.05	-.07
MOBILITY	II,I	.12	.02	.27	-.02	.04	-.05
MOBILITY	II,II	.14	.02	.15	.04	.07	.22
SOCIAL	II,I	-.11	.16	.13	.32*	-.16	-.08
SOCIAL	II,II	-.15	.08	.06	-.01	.03	.07
PHYSSIP	II,I	.14	.19	.23	.17	.03	.12
PHYSSIP	II,II	.18	.05	-.02	.02	.03	-.04
PSYCHSIP	II,I	-.03	.16	.10	.33*	-.18	.07
PSYCHSIP	II,II	-.05	.09	.14	-.05	.06	.09
TOTSIP	II,I	.02	.18	.14	.36*	-.16	.08
TOTSIP	II,II	.05	.06	.07	-.01	-.01	.01

		HbA1c	Ca9	AFFECT	WELLBEING
HbA1c	II,I	1.00	.26	.02	.16
HbA1c	II,II	1.00	.24*	.04	.15
Ca9	II,I	.26	1.00	.64**	.91**
Ca9	II,II	.24*	1.00	.74**	.94**
AFFECT	II,I	.02	.64**	1.00	.89**
AFFECT	II,II	.04	.74**	1.00	.93**
WELLBEING	II,I	.16	.91**	.89**	1.00
WELLBEING	II,II	.15	.94**	.93**	1.00

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		HbA1c	Ca9	AFFECT	WELLBEING
IHLC	II,I	.06	.11	.06	.09
IHLC	II,II	.00	.17	.00	.11
POLC	II,I	.08	.21	-.00	.13
POLC	II,II	.12	.02	-.02	.01
CHLC	II,I	.32*	-.03	-.41**	-.24
CHLC	II,II	.05	-.18	-.15	-.18
SES	II,I	-.04	.43**	.77**	.66**
SES	II,II	-.07	.47**	.59**	.57**
NPPATHY	II,I	-.00	-.04	-.10	-.08
NPPATHY	II,II	.22	-.16	-.18	-.17
RTPATHY	II,I	--	--	--	--
RTPATHY	II,II	-.10	.01	-.17	-.09
HYP	II,I	-.32*	-.23	-.42**	-.36*
HYP	II,II	-.23*	-.29*	-.34**	-.33**
DEP	II,I	-.35*	-.41**	-.66**	-.59**
DEP	II,II	-.06	-.50**	-.74**	-.65**
DEN	II,I	.02	.15	.24	.23
DEN	II,II	-.02	.13	.22*	.18
INP	II,I	-.09	-.27	-.44**	-.40*
INP	II,II	.17	-.21	-.39**	-.31*
ALN	II,I	-.14	-.34*	-.20	-.30*
ALN	II,II	.11	-.20	-.41**	-.32**
PID	II,I	-.06	-.15	-.46**	-.33*
PID	II,II	-.14	-.21	-.30*	-.26*

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		HbA1c	Ca9	AFFECT	WELLBEING
ANX	II,I	-.19	-.28	-.58**	-.47**
ANX	II,II	-.11	-.34**	-.36**	-.37**
THD	II,I	-.01	-.04	-.50**	-.30*
THD	II,II	-.14	-.28*	-.36**	-.35**
IME	II,I	-.15	-.33*	-.41**	-.42**
IME	II,II	.18	-.11	-.37**	-.25*
SOI	II,I	-.17	-.15	-.25	-.23
SOI	II,II	.07	-.06	-.33**	-.20
SDP	II,I	-.27	-.33*	-.62**	-.52**
SDP	II,II	.07	-.36**	-.46**	-.44**
DEV	II,I	-.28	-.22	-.60**	-.44**
DEV	II,II	-.02	-.15	-.39**	-.28*
AMBUL	II,I	-.03	-.04	.04	-.01
AMBUL	II,II	.10	-.16	-.21	-.19
ALERT	II,I	-.12	-.01	-.37	-.20
ALERT	II,II	.23*	-.28	-.41**	-.37**
COMMUN	II,I	-.09	-.15	-.48**	-.34*
COMMUN	II,II	.04	-.02	-.19	-.11
WORK	II,I	-.28	.02	-.35*	-.18
WORK	II,II	.06	-.13	-.14	-.15
RECREAT	II,I	-.16	.01	-.32	-.17
RECREAT	II,II	.06	.24*	.12	.20
EATING	II,I	.27	-.09	-.10	-.10
EATING	II,II	-.16	.02	.07	.06

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		HbA1c	Ca9	AFFECT	WELLBEING
SLEEP	II,I	-.22	-.31*	-.59**	-.50**
SLEEP	II,II	.02	-.20	-.46**	.93**
EMOTION	II,I	-.06	.02	-.54**	-.28
EMOTION	II,II	-.09	-.32**	-.43**	-.40**
BODYCARE	II,I	-.03	-.26	-.25	-.28
BODYCARE	II,II	-.12	-.23	-.22*	-.24*
HOME	II,I	-.26	-.30*	-.43**	-.41**
HOME	II,II	.04	-.35**	-.34**	-.37**
MOBILITY	II,I	-.06	-.37*	-.30*	-.37*
MOBILITY	II,II	.04	-.17	-.38**	-.29*
SOCIAL	II,I	-.11	-.05	-.59**	-.35*
SOCIAL	II,II	.10	-.10	-.25*	-.19
PHYSSIP	II,I	-.05	-.27	-.21	-.27
PHYSSIP	II,II	-.01	-.22*	-.29*	-.27*
PSYCHSIP	II,I	-.12	-.03	-.55**	-.31*
PSYCHSIP	II,II	.12	-.24*	-.41**	-.35**
TOTSIP	II,I	-.15	-.11	-.57**	-.37*
TOTSIP	II,II	.06	-.24*	-.38**	-.33**

		NPPATHY	RTPATHY	IHLC	POLC	CHLC	SES
NPPATHY	II,I	1.00	--	.00	.21	.14	-.29
NPPATHY	II,II	1.00	.19	.11	-.09	-.20	-.16
RTPATHY	II,I	--	1.00	--	--	--	--
RTPATHY	II,II	.19	1.00	-.14	-.05	-.08	-.02

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		NPPATHY	RTPATHY	IHLC	POLC	CHLC	SES
IHLC	II,I	.00	--	1.00	-.14	.02	.11
IHLC	II,II	.11	-.14	1.00	-.03	-.25*	.09
POLC	II,I	.21	--	-.14	1.00	.49**	.07
POLC	II,II	-.09	-.05	-.03	1.00	.18	-.18
CHLC	II,I	.14	--	.02	.49**	1.00	-.41**
CHLC	II,II	-.20	-.08	-.25*	.18	1.00	-.30*
SES	II,I	-.29	--	.11	.07	-.41**	1.00
SES	II,II	-.16	-.02	.09	-.18	-.30*	1.00
HYP	II,I	.39*	--	-.01	-.04	.10	-.39*
HYP	II,II	-.02	.19	.02	.17	.22*	-.29*
DEP	II,I	-.07	--	-.10	-.12	.22	-.56**
DEP	II,II	.27*	.01	.22	.18	.03	-.59**
DEN	II,I	.03	--	-.00	.33*	-.17	.35*
DEN	II,II	-.00	.06	-.16	.19	-.06	.27*
INP	II,I	.16	--	-.02	.05	.41**	-.41**
INP	II,II	.24*	.12	-.16	.12	.10	-.34**
ALN	II,I	-.11	--	-.16	.06	.11	-.08
ALN	II,II	.09	.24*	-.00	-.01	.17	-.26*
PID	II,I	.02	.02	-.14	.13	.55**	-.45**
PID	II,II	.12	.10	.02	.09	.22	-.32**
ANX	II,I	.23	--	-.20	-.07	.26	-.56**
ANX	II,II	-.03	-.20	-.06	.13	.32**	-.50**
THD	II,I	.17	--	-.16	.00	.46**	-.56**
THD	II,II	-.22	-.09	-.12	.22	.34**	-.34**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		NPPATHY	RTPATHY	IHLC	POLC	CHLC	SES
IME	II,I	.38*	--	-.06	-.13	.29	-.47**
IME	II,II	.16	.08	.21	.05	-.07	-.35**
SOI	II,I	.10	--	.06	.00	.38	-.42**
SOI	II,II	.12	.15	.10	.23**	.02	-.32**
SDP	II,I	.05	--	-.34*	-.08	.25	-.73**
SDP	II,II	.31*	.24*	-.04	.27*	.19	-.60**
DEV	II,I	.05	--	-.19	.08	.38	-.44**
DEV	II,II	.03	.19	.08	.19	.10	-.37**
AMBUL	II,I	.15	--	-.31	.27	-.02	.05
AMBUL	II,II	-.03	.17	-.06	.37**	.06	-.33**
ALERT	II,I	.02	--	-.03	-.07	.35*	-.36*
ALERT	II,II	.10	.12	-.07	.19	.16	-.49**
COMMUN	II,I	-.09	--	-.17	.16	.45**	-.30*
COMMUN	II,II	-.22	.16	-.08	.18	.10	-.19
WORK	II,I	.06	--	.08	.15	.29*	-.45**
WORK	II,II	.04	.07	.09	.03	-.20	-.10
RECREAT	II,I	-.02	--	-.02	-.12	.00	-.39*
RECREAT	II,II	-.16	.13	.10	.16	-.05	-.04
EATING	II,I	.01	--	-.24	.18	.36*	-.06
EATING	II,II	-.04	.04	-.04	-.12	.07	-.03
SLEEP	II,I	.22	--	.02	-.20	.20	-.62**
SLEEP	II,II	.05	.05	.10	.14	.17	-.57**
EMOTION	II,I	-.02	--	-.03	-.02	.38*	-.58**
EMOTION	II,II	.06	.14	.05	.02	.14	-.38**

* $p \leq .05$
** $p \leq .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		NPPATHY	RTPATHY	IHLC	POLC	CHLC	SES
BODYCARE	II,I	-.10	--	.01	-.05	.38*	-.28**
BODYCARE	II,II	-.06	.13	-.02	.24*	.03	-.24*
HOME	II,I	-.17	--	.03	-.32*	-.07	-.45**
HOME	II,II	-.16	.03	-.15	.16	.29*	-.35**
MOBILITY	II,I	-.09	--	-.09	-.10	.04	-.21
MOBILITY	II,II	.08	.43**	-.06	.18	.08	-.22*
SOCIAL	II,I	.13	--	.03	-.01	.31*	-.52**
SOCIAL	II,II	.05	.14	.05	.02	.02	-.38**
PHYSSIP	II,I	-.03	--	-.16	.07	.21	-.20
PHYSSIP	II,II	-.02	.24*	-.05	.29*	.06	-.29*
PSYCHSIP	II,I	-.13	--	-.05	.06	.45**	-.50**
PSYCHSIP	II,II	.03	.18	.01	.13	.12	-.49**
TOTSIP	II,I	-.09	--	-.07	.04	.43**	-.55**
TOTSIP	II,II	-.02	.19	.00	.22*	.10	-.46**

		AMBUL	ALERT	COMMUN	WORK	RECREAT
AMBUL	II,I	1.00	.23	.29*	.04	.16
AMBUL	II,II	1.00	.31**	.40**	.40**	.58**
ALERT	II,I	.23	1.00	.66**	.59**	.73**
ALERT	II,II	.31**	1.00	.38**	.16	.17
COMMUN	II,I	.29*	.66**	1.00	.35*	.32*
COMMUN	II,II	.40**	.38**	1.00	.19	.36**
WORK	II,I	.04	.59**	.35*	1.00	.67**
WORK	II,II	.40**	.16	.19	1.00	.42**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		AMBUL	ALERT	COMMUN	WORK	RECREAT
RECREAT	II,I	.16	.73**	.32*	.67**	1.00
RECREAT	II,II	.58**	.17	.36**	.42**	1.00
EATING	II,I	.16	-.10	.12	-.16	-.16
EATING	II,II	.08	.22*	.28*	-.10	.18
SLEEP	II,I	.15	.61**	.50**	.39*	.50**
SLEEP	II,II	.35**	.37**	.38**	.35**	.23*
EMOTION	II,I	.05	.78**	.62**	.77**	.65**
EMOTION	II,II	.33**	.50**	.17	.43**	.24*
BODYCARE	II,	.36*	.56**	.47**	.28*	.14
BODYCARE	II,II	.71**	.20	.34**	.46**	.44**
HOME	II,I	.34*	.56**	.60**	.45**	.55**
HOME	II,II	.48**	.38**	.28**	.22*	.36**
MOBILITY	II,I	.39*	.08	.58**	-.10	.01
MOBILITY	II,I	.51**	.27*	.39**	.39**	.37**
SOCIAL	II,I	.09	.80**	.76**	.60**	.69**
SOCIAL	II,II	.45**	.53**	.37**	.47**	.46**
PHYSSIP	II,I	.77**	.43**	.56**	.14	.15
PHYSSIP	II,II	.86**	.29*	.42**	.48**	.53**
PSYCHSIP	II,I	.13	.92**	.77**	.71**	.71**
PSYCHSIP	II,II	.48**	.83**	.55**	.41**	.40**
TOTSIP	II,I	.24	.91**	.78**	.72**	.74**
TOTSIP	II,II	.75**	.60**	.55**	.60**	.61**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		EATING	SLEEP	EMOTION	BODYCARE	HOME
AMBUL	II,I	.16	.15	.05	.36*	.34*
AMBUL	II,II	.08	.35**	.33**	.71**	.48**
ALERT	II,I	-.10	.61**	.78**	.56**	.56**
ALERT	II,II	.22*	.37**	.50**	.20	.38**
COMMUN	II,I	.12	.50**	.62**	.47**	.60**
COMMUN	II,II	.28*	.38**	.17	.34**	.28**
WORK	II,I	-.16	.39*	.77**	.28*	.45**
WORK	II,II	-.10	.35**	.43**	.46**	.22*
RECREAT	II,I	-.16	.50**	.65**	.14	.55**
RECREAT	II,II	.18	.23*	.24*	.44**	.36**
EATING	II,I	1.00	-.12	-.16	.17	-.13
EATING	II,II	1.00	.07	-.06	.00	.00
SLEEP	II,I	-.12	1.00	.57**	.37*	.71**
SLEEP	II,II	.07	1.00	.56**	.49**	.36**
EMOTION	II,I	-.16	.57**	1.00	.38*	.53**
EMOTION	II,II	-.06	.56**	1.00	.53**	.48**
BODYCARE	II,I	.17	.37*	.38*	1.00	.44**
BODYCARE	II,II	.00	.49**	.53**	1.00	.53**
HOME	II,I	-.13	.71**	.53**	.44**	1.00
HOME	II,II	.00	.26*	.48**	.53**	1.00
MOBILITY	II,I	.34*	.31*	.02	.37*	.61**
MOBILITY	II,II	-.14	.53**	.54**	.67**	.50**
SOCIAL	II,I	-.21	.70**	.87**	.26	.65**
SOCIAL	II,II	.16	.50**	.63**	.53**	.32**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		EATING	SLEEP	EMOTION	BODYCARE	HOME
PHYSSIP	II,I	.27	.36*	.23	.81**	.58**
PHYSSIP	II,II	-.01	.52**	.53**	.95**	.58**
PSYCHSIP	II,I	-.10	.66**	.92**	.43**	.60**
PSYCHSIP	II,I	.20	.58**	.75**	.51**	.47**
TOTSIP	II,I	.02	.74**	.88**	.52**	.71**
TOTSIP	II,II	.13	.66**	.73**	.80**	.62**

		MOBILITY	SOCIAL	PHYSSIP	PSYCHSIP	TOTSIP
AMBUL	II,I	.39	.09	.77**	.13	.24
AMBUL	II,II	.51**	.45**	.86**	.48**	.75**
ALERT	II,I	.08	.80**	.43**	.92**	.91**
ALERT	II,II	.27*	.53**	.29*	.83**	.60**
COMMUN	II,I	.58**	.76**	.56**	.77**	.78**
COMMUN	II,II	.39**	.37**	.42**	.55**	.55**
WORK	II,I	-.10	.60**	.14	.71**	.72**
WORK	II,II	.39**	.47**	.48**	.41**	.60**
RECREAT	II,I	.01	.69**	.15	.71**	.74**
RECREAT	II,II	.37**	.46**	.53**	.40**	.61**
EATING	II,I	.34*	-.21	.27	-.10	-.02
EATING	II,II	-.14	.16	-.01	.20	.13
SLEEP	II,I	.31*	.70**	.36*	.66**	.74**
SLEEP	II,II	.53**	.50**	.52**	.58**	.66**
EMOTION	II,I	.02	.87**	.23	.92**	.88**
EMOTION	II,II	.54**	.63**	.53**	.75**	.73**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		MOBILITY	SOCIAL	PHYSSIP	PSYCHSIP	TOTSIP
BODYCARE	II,I	.37*	.27	.81**	.43**	.52**
BODYCARE	II,II	.67**	.53**	.95**	.51**	.80**
HOME	II,I	.61***	.65**	.58**	.60**	.71**
HOME	II,II	.50**	.32**	.58**	.47**	.62**
MOBILITY	II,I	1.00	.17	.69**	.17	.29*
MOBILITY	II,II	1.00	.36**	.79**	.47**	.70**
SOCIAL	II,I	.17	1.00	.23	.97**	.94**
SOCIAL	II,II	.36**	1.00	.47**	.87**	.79**
PHYSSIP	II,I	.69**	.23	1.00	.34*	.48**
PHYSSIP	II,II	.79**	.47**	1.00	.56**	.86**
PSYCHSIP	II,I	.17	.97**	.34*	1.00	.98**
PSYCHSIP	II,II	.47**	.87**	.56**	1.00	.87**
TOTSIP	II,I	.29*	.94**	.48**	.98**	1.00
TOTSIP	II,II	.70**	.79**	.86**	.87**	1.00

		AMBUL	ALERT	COMMUN	WORK	RECREAT
HYP	II,I	.26	.47**	.40*	.37*	.45**
HYP	II,II	.51**	.25*	.38**	.27*	.37**
DEP	II,I	.12	.60**	.71**	.57**	.42**
DEP	II,II	.33**	.51**	.31*	.26*	.12
DEN	II,I	.05	-.46**	-.21	-.29*	-.43**
DEN	II,II	.03	-.16	.02	-.27*	-.06
INP	II,I	.25	.51**	.44**	.53**	.35*
INP	II,II	.31*	.32**	.27*	.21	.10

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		AMBUL	ALERT	COMMUN	WORK	RECREAT
ALN	II,I	.18	.15	.41**	.11	-.10
ALN	II,II	.11	.34**	.37**	.13	.11
PID	II,I	-.01	.51**	.58**	.36*	.22
PID	II,I	.01	.32**	.03	-.08	-.05
ANX	II,I	.19	.47**	.54**	.50**	.40*
ANX	II,II	.06	.23*	-.17	-.19	-.09
THD	II,I	.01	.58*	.55**	.66**	.55**
THD	II,II	.01	.25*	.13	-.10	-.17
IME	II,I	.17	.55**	.36*	.44**	.40**
IME	II,II	.09	.44**	.18	.26*	.11
SOI	II,I	.08	.21	.46**	.34*	.09
SOI	II,II	.09	.32**	.35**	-.07	.12
SDP	II,I	.15	.31*	.39*	.45**	.25
SDP	II,II	.11	.40**	-.01	.11	-.19
DEV	II,I	.16	.61**	.75**	.43**	.38**
DEV	II,II	.18	.42**	.27**	.12	.21

		EATING	SLEEP	EMOTION	BODYCARE	HOME
HYP	II,I	-.09	.55**	.53**	.09	.43**
HYP	II,II	.10	.37**	.37**	.50**	.27*
DEP	II,I	-.14	.72**	.71**	.48**	.72**
DEP	II,II	.08	.57**	.48**	.36**	.36**
DEN	II,I	.14	-.23	-.41**	-.20	-.18
DEN	II,II	-.18	-.09	-.19	.05	-.04

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		EATING	SLEEP	EMOTION	BODYCARE	HOME
INP	II,I	.18	.42**	.48**	.32**	.37**
INP	II,II	.25*	.29*	.35**	.29*	.22*
ALN	II,I	-.11	.28	.13	.40*	.33*
ALN	II,II	.39**	.38**	.33**	.35**	.26*
PID	II,I	-.13	.54**	.52**	.31*	.34*
PID	II,II	.33**	.14	.39**	.16	.27*
ANX	II,I	.01	.58**	.61**	.19	.52**
ANX	II,II	.11	.21	.14	.02	-.07
THD	II,I	-.05	.44**	.75**	.14	.47**
THD	II,II	.18	.25*	.15	.25	.21
IME	II,I	-.02	.47**	.49**	.31*	.34*
IME	II,II	.26*	.31*	.29*	.22*	.12
SOI	II,I	.02	.59**	.29*	.37*	.54**
SOI	II,II	.22*	.20	.11	.07	.18
SDP	II,I	.02	.58**	.52**	.31*	.42**
SDP	II,II	.00	.40**	.29*	.09	.07
DEV	II,I	-.08	.61**	.69**	.23	.44**
DEV	II,II	.24*	.52**	.41**	.46**	.15

		MOBILITY	SOCIAL	PHYSSIP	PSYCHSIP	TOTSIP
HYP	II,I	.02	.60**	.17	.52**	.54**
HYP	II,II	.38**	.25*	.53**	.37**	.52**
DEP	II,I	.38*	.72**	.43**	.75**	.79**
DEP	II,II	.38**	.35**	.40**	.53**	.54**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		MOBILITY	SOCIAL	PHYSSIP	PSYCHSIP	TOTSIP
DEN	II,I	.15	-.43**	-.04	-.43**	-.40*
DEN	II,II	.05	-.20	.05	-.19	-.13
INP	II,I	.09	.50**	.31*	.54**	.58**
INP	II,II	.38**	.37**	.36**	.42**	.43**
ALN	II,I	.28	.18	.38*	.22	.25
ALN	II,II	.37**	.39**	.32**	.46**	.44**
PID	II,I	.11	.59**	.19	.62**	.60**
PID	II,II	.06	.34**	.10	.37**	.25*
ANX	II,I	.18	.64**	.24	.59**	.61**
ANX	II,II	-.11	.02	-.00	.11	.04
THD	II,I	.10	.71**	.11	.72**	.70**
THD	II,II	.08	.10	.16	.21	.18
IME	II,I	-.02	.50**	.24	.50**	.52**
IME	II,II	.22	.38**	.20	.45**	.38**
SOI	II,I	.49**	.30*	.38*	.35*	.44**
SOI	II,II	.15	.07	.11	.25*	.21
SDP	II,I	.25	.37*	.31*	.49**	.56**
SDP	II,II	.15	.17	.12	.30*	.23*
DEV	II,I	.19	.75**	.26	.76**	.74**
DEV	II,II	.36**	.36**	.40**	.48**	.49**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		HYP	DEP	DEN	INP	ALN	PID
HYP	II,I	1.00	.53**	-.40*	.45**	.12	.27
HYP	II,II	1.00	.47**	.01	.25*	.20	.17
DEP	II,I	.53**	1.00	-.28	.45**	.53**	.63**
DEP	II,II	.47**	1.00	-.25	.46**	.57**	.41**
DEN	II,I	-.39*	-.28	1.00	-.59**	-.18	-.38*
DEN	II,II	.01	-.25*	1.00	-.25*	-.35**	-.25*
INP	II,I	.44**	.45**	-.59**	1.00	.31*	.44**
INP	II,II	.25*	.46**	-.25*	1.00	.56*	.39**
ALN	II,I	.12	.53**	-.18	.31*	1.00	.57**
ALN	II,II	.20	.56**	-.35**	.56**	1.00	.44**
PID	II,I	.27	.63**	-.38*	.44**	.57**	1.00
PID	II,II	.17	.41**	-.25	.39**	.44**	1.00
ANX	II,I	.62**	.63**	-.60**	.70**	.31*	.53**
ANX	II,II	.24*	.36**	-.30*	.25*	.18	.39**
THD	II,I	.43**	.57**	-.48**	.52**	.15	.61**
THD	II,II	.18	.45**	-.17	.22*	.47**	.56**
IME	II,I	.52**	.41**	-.66**	.80**	.23	.36*
IME	II,II	.12	.52**	-.52**	.61**	.69**	.44**
SOI	II,I	.19	.52**	-.03	.26	.29*	.55**
SOI	II,II	.47**	.50**	.15	.32**	.36**	.32**
SDP	II,I	.31*	.68**	-.25	.40*	.30*	.49**
SDP	II,II	.22*	.52**	-.08	.37**	.38**	.42**
DEV	II,I	.61**	.75**	-.43**	.52**	.49**	.72**
DEV	II,II	.53**	.57**	-.06	.32**	.54**	.45**

* $p < .05$
** $p < .01$

CORRELATIONAL TABLES
FOR TYPE II, ONSET I AND TYPE II, ONSET II GROUPS

		ANX	THD	IME	SOI	SDP	DEV
HYP	II,I	.62**	.73**	.52**	.19	.31*	.61**
HYP	II,II	.24*	.18	.12	.47**	.22	.39**
DEP	II,I	.63**	.57**	.41**	.52**	.68	.75**
DEP	II,II	.36**	.45**	.52**	.50**	.52**	.56**
DEN	II,I	-.60**	-.48**	-.66**	-.03	-.25	-.43**
DEN	II,II	-.30*	.52**	-.52**	.15	-.08	.44**
INP	II,I	.70**	.52**	.80**	.26	.40*	.52**
INP	II,II	.25*	.50**	.61**	.32**	.36**	.32**
ALN	II,I	.31*	.53**	.23	.29*	.30*	.49**
ALN	II,II	.18	.52**	.69**	.36**	.38**	.42**
PID	II,I	.53**	.61**	.36*	.55**	.49**	.72**
PID	II,II	.39**	.57**	-.44**	.32**	.42**	.45**
ANX	II,I	1.00	.69**	.69**	.35*	.60**	.71**
ANX	II,II	1.00	.50**	.25*	.25*	.39**	.40**
THD	II,I	.69**	1.00	.55**	.40*	.53**	.69**
THD	II,II	.50**	1.00	.37**	.34**	.45**	.53**
IME	II,I	.69**	.55**	1.00	.15	.42**	.59**
IME	II,II	.25*	.37**	1.00	.32**	.44**	.44**
SOI	II,I	.35*	.40*	.15	1.00	.43**	.30*
SOI	II,II	.25*	.34**	.32**	1.00	.49**	.54**
SDP	II,I	.60**	.53**	.42**	.43**	1.00	.63**
SDP	II,II	.39**	.45**	.44**	.49**	1.00	.42**
DEV	II,I	.71**	.69**	.59**	.30*	.63**	1.00
DEV	II,II	.40**	.53**	.44**	.54**	.42**	1.00

* $p < .05$
** $p < .01$